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GASTRIC ULCER - ITS TREATMENT WITH GELATIN

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by

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## PREFACE.

Every Practitioner of Medicine who is actively engaged in practice must often ask himself the questions - Do we advance in our knowledge? Do we improve in our treatment?

In our daily life we are immersed in the details of individual cases. We are embarrassed by the variety of the facts placed before us and by the very nearness of our contact our judgment may be obscured. Regarding the great amount of laborious research which this subject has received in this and other countries, it might be said there is no room left for further investigation or improvement. In holding the position of House Physician of a large provincial Hospital it fell to my lot to see a large number of cases of this interesting disease. Although in treating of the clinical history of this disorder I have been for the most part moving over familiar ground, yet we are told "the old order changeth yielding place to new" and the truth of this doctrine is attested continually.

The task I have assigned myself is to consider a new method of treatment. I have ventured to submit in this thesis a number of cases of gastric ulcer treated by a new and, to a certain extent, experimental method with I hope to show a considerable measure of success.

## Ulcer of the Stomach.

### History.

Ulcer of the stomach was first recognised as a definite and well characterised disease by Cruveilhier and was described by him in the year 1829. We are indebted to him for the first exact anatomical description of this lesion and also for the first careful account of its characteristic symptomatology and of a number of measures for its treatment. Previous to Cruveilhier's description/<sup>although</sup> a few reports of cases of ulcer of the stomach had been published, the points of difference between this and other forms of ulceration of the stomach, e.g. the carcinomatous, were unknown. Following Cruveilhier's lead many authors have added classical investigations on this subject to the literature and as science has elaborated and made more perfect the methods of research, scarcely a year has passed without some contribution being made either to the elucidation of its etiology or to its treatment.

### Etiology.

Although gastric ulcer is a disease frequently encountered by every physician, its exact incidence is difficult to determine. Byrom Bramwell has pointed out that post-mortem record statistics may lead one astray/



astray from the<sup>true</sup>/facts due to the possibility of all active ulcers not being found, cicatrices of ulcers being overlooked, of ulcers healed and leaving no visible scar, or of cicatrices due to other causes apart from gastric ulcer being mistaken for these. Likewise statistics based on clinical observations are unreliable due to the difficulty in many cases of diagnosis.

Statistics compiled from records of post-mortem examinations vary considerably according to geographical distribution. In 200 autopsies Lebert found one case. At the other extreme Grünfeld gave a percentage of twenty.

In the United States and Canada, Campbell Howard (1) has recorded 144 cases of ulcer out of 10,841 autopsies, or 1.3%, whereas in London 4.6% of 10,000 autopsies were cases of gastric ulcer (Fenwick), and on the Continent 5% was found.

Clinical statistics also show similar variations. Lebert out of 41,688 medical admissions found 0.66% to be cases of gastric ulcer in Breslau and Zurich over a period of twenty years. In ten years, in the Charité Hospital, Berlin, the percentage was 1.33 of 42,219 admissions. In ten years in The London and The London Temperance Hospitals 0.82 per cent of 45,712 admissions were noted to be cases of gastric ulcer; while in Edinburgh in eight years in 35,692 admissions/

admissions it occurred in 2.2% and in 7,765 outpatients it was met with in 0.09%, giving a combined incidence of 2.02% (Byrom Bramwell).

Sex. Most authorities give the incidence as being greater in females than in males, but Mayo and others state the reverse to be the rule. The generally accepted ratio is two females to one male.

Age seems to be a determining factor in the development of ulcer. Before puberty gastric ulcer is rare, although cases have been recorded. In females gastric ulcer is most frequently met with between the ages of twenty and thirty, whereas in males the age is thirty to fifty. Osler states that symptoms of gastric ulcer have come on in the newly born and various authors record cases in the aged.

Geographical distribution. Place seems to have its influence. Thus ulcer is more frequent in Europe than in America and more so in North than South Europe, and less frequent in France than in adjacent countries. Edinburgh has a higher statistical record than London.

Occupation. Although gastric ulcer is apparently more frequent in those engaged in domestic service, these are generally young chlorotic females and probably sex and age have more to do with its development than occupation.

Associated Diseases. By some gastric ulcer is believed to/

to occur more frequently in certain disease conditions. Thus chlorosis and gastric ulcer are frequently met with together. The Fenwicks found it in 72% of acute cases of ulcer. It has also been observed to occur during the course of valvular heart disease, chronic nephritis, septicaemia and pyaemia, malaria and scurvy; also in tuberculosis and syphilis and in amenorrhoea.

Traumatism. Ulcer of the stomach may very probably follow simple external violence under certain conditions, but there always remains the doubt whether the symptoms are due primarily to the injury or only become manifest after it, the ulcer having been present before.

Heredity. It is extremely doubtful whether heredity has any bearing upon the incidence of ulcer of the stomach and most authors avoid expressing an opinion.

#### Pathogenesis.

The causation of gastric ulcer and our knowledge of it are still very indefinite although a large number of theories have been advanced and experiments performed in endeavours towards its elucidation, and thus many diverse views are held as to its pathogenesis. In all probability this indefinite position as to the exact nature of the cause of gastric ulcer can be explained on the ground that it is not always due to one and the same cause and that in some cases there may be several factors working together either directly or/

or indirectly in its production. (2).

The fact that these ulcers are found only in the stomach or in its immediate neighbourhood (e.g., in the duodenum) points to the gastric juice as playing an important rôle in their production along with some as yet unknown devitalising local condition which allows ~~on~~ its eroding action to come into play.

Hunter was of the opinion that there was loss of some vital principle inherent in the cells of the mucosa. Claude Bernard (3), following upon experiments which showed that living tissues were digested if put into the stomach during the process of digestion and which thus undermined Hunter's theory, argued that the mere fact of life being present did not of itself protect the stomach from autodigestion and suggested that in reality a constant destruction of the superficial layers of the mucosa was going on and that the deeper layers were protected by the rapid and efficient proliferative powers of the superficial cells and that some depression of this proliferative power was accountable for the occurrence of ulceration. Pavy corroborated Bernard's experiments, but advanced the theory of a defective circulation of alkaline blood in the part, thus allowing the inefficiently neutralised hydrochloric acid to have a destructive action on the deeper layers of the wall of the stomach.

Various/



Various theories as to disturbances of circulation, inflammation, bacteria and their toxins, and trauma have been advanced as causing necrobiosis of the tissues. Stockton (4) attributed gastric ulcer to the result of a neurotrophic change, and Weinland and Cooke to a loss of specific protective anti-ferments from the mucosal cells.

Although a large number of lesions very similar in structural peculiarities to ulcer of the stomach in humans have been experimentally produced in animals, the ulcer thus produced has lacked the tendency to extend or its intractability to healing.

Disturbances of Circulation. Virchow attributed the lesion to a localised ischaemic necrosis following arterial obstruction due to embolism. This view obtained wide credence and was corroborated by the experiments of Cohnheim (5). Although it may hold good for some cases, the production of the great majority of gastric ulcers cannot be explained in this way because it is only very few cases that show lesions from which emboli might originate and also other organs rarely show evidence of infarction.

Arterial thrombosis as a cause of circumscribed necrosis is also open to doubt as this occurs in subjects whose arterial walls are diseased, and as a rule at the age at which gastric ulcer occurs it is extremely rare to find degenerative changes in the blood/



blood vessels. But in middle aged persons or in younger persons afflicted with syphilis or tuberculosis, atheroma of the coronary artery with secondary thrombosis may be a factor to be counted with.

Recent investigators are inclined to uphold Virchow's view. Wilkie (6), as the result of his experiments on animals, arrives at the following conclusions:- (a) Thrombosis of the veins of the omentum is readily produced by mechanical, thermal and bacterial agencies. (b) From thrombosed omental veins emboli may frequently separate, probably owing to the periodic vaso-dilatation and contraction associated with the ingestion of food. (c) Emboli from veins in the omentum may, under certain circumstances, be carried up gastric veins and become impacted in the venous plexus of the submucous coat of the stomach and determine gastric ulcer.

Fenwick and Müller produced intense congestion of the veins of the gastric mucosa with haemorrhages which frequently became ulcers, by occlusion of the portal vein. These ulcers differed markedly, however, from true gastric ulcer.

Thus we see that although, experimentally, interference with the circulation of the blood in a circumscribed area of the gastric mucosa can produce ulcers, these ulcers do not answer in all characteristics to the gastric ulcer in human beings and post-mortem evidence/

evidence of the necessary special conditions for their production is lacking in cases of gastric ulcer.

Trauma. Here we have three classes of agents - Mechanical, chemical and thermal.

Mechanical. In a few cases clinical observation has shown that ulceration may follow mechanical injury to the stomach, e.g. from a blow on the epigastrium. Ulcers have also been produced experimentally in dogs in this way. Experiment has also proved that direct injury to the gastric mucosa does not lead to the formation of an ulcer. Frequently when washing out the stomach large pieces of mucous membrane have been accidentally abraded, but the resulting lesion has been found to be very tractable and followed by no serious results.

Chemical traumatism due to the action of corrosives, both acid and alkaline, is a fairly frequent accident, but this cause will only explain very few of all cases of gastric ulcer.

Thermal. The proneness with which cooks are liable to suffer from gastric ulcer has been put down to the habit of consuming very hot food. Decker produced ulceration in the stomach of a dog in this way, which had all the characteristics of an acute ulcer. Here again this could be only a very infrequent cause.

Neurotrophic/

Neurotrophic Theory. Some authors hold that through loss of nervous control over the various functions of the stomach, such as that of secretion or vascularity, the nervous system may play a part in the production of gastric ulcer. Experiments and also clinical observations have shown that injury to and disease of the central nervous system are responsible for minute haemorrhages or haemorrhagic erosions in the mucosa but it is doubtful whether these ever develop into gastric ulcers. Yzeran (7), by section of the vagus below the diaphragm, produced gastric ulcers in rabbits as also did Talma by the faradic stimulation of the vagus. Donati, on repeating Yzeran's experiments, failed to produce ulcers but observed an increased acid secretion of the gastric juice. Stockton's theory was that there was some distinct and persevering nerve perturbation as the origin of low tissue resistance allowing the gastric juice to become destructive.

Gastritis. Among early writers (Cruveilhier and Abercrombie) gastritis was believed to be the essential cause of gastric ulcer. This theory was abandoned in favour of Virchow's circulatory theory, but has recently been revived by Continental writers.

Dr. Soltan Fenwick holds the view that an inflammatory condition of the lymphoid follicles of the stomach which are always more numerous at the pylorus/

pylorus and lesser curvature may sometimes be the origin of gastric ulcer. These follicles in certain diseases, e.g. gastritis and some infective fevers, are acutely inflamed, rapidly break down and form numerous small lesions surrounded by congested areas. C. H. Miller, from examining a large number of stomachs agrees with Dr. Fenwick's views and believes that contraction around these lesions is incomplete due to inflammatory changes, thus leaving the lesion open to the action of the gastric secretion.

Although all the preceding conditions are capable of producing ulceration of the stomach due to interference with the vitality of the stomach wall they are not by themselves responsible for the typical ulcer as it occurs in human beings, and therefore other factors must be at work which confer upon the lesion its characteristic tendency to progression and its reluctance to heal. These factors may be found under the following headings:-

Situation. Ulcers which have been produced experimentally have been shown to heal less quickly when situated near the lesser curvature or pylorus than those on the greater curvature or at the fundus of the stomach. The explanation given of this is that in these regions of the stomach the peristaltic movements are more vigorous and thus the rest essential for the healing process is wanting.

The/



The Gastric Secretion. The presence of free hydrochloric acid in the gastric juice has been considered as of great importance in the development of gastric ulcer. Riegel regarded hyperacidity as primary to the lesion and characteristic of all cases of gastric ulcer. In support of this view Matthes and Cohnheim showed that lesions, which under ordinary circumstances healed quickly, were slow in healing if hyperacidity were produced artificially. On the other hand, many authors regard hyperchlorhydria as an inconstant feature and, when present, as being secondary to the lesion. Thus there is diversity of opinion on this point and cases have been recorded in which hyperchlorhydria was absent altogether.

Alterations in the blood. Although chlorosis is a frequent associated condition with gastric ulcer, there is some difference of opinion as to the exact relationship they bear to one another. Due to the abnormal composition of the blood in this condition, chlorosis has been suggested as a cause of thrombosis or of haemorrhages into the submucous coat and thus in either of these ways as being a primary factor in the production of an ulcer.

Another and much more tenable view is that chlorosis, either by depressing the recuperative power of the tissues or by altering the composition of the gastric secretion (Riegel (8)) might be of great/



great importance in producing gastric ulcer.

Quincke and Daettwyler, after experimenting on dogs, formed the opinion that any slight injury to the stomach in an anaemic person may develop into an ulcer and that ulcers are retarded from healing by anaemia.

In support of Riegel's view that in chlorosis there is frequently hyperacidity of the gastric juice, Grüne found hyperacidity in all the cases he examined, Schätzell in 73 per cent and Cantu in nearly all his series of cases. Thus the combination of anaemia and hyperacidity may have an important bearing upon the development of gastric ulcer ~~either~~ by depressing the resistive powers of the tissues and at the same time preventing the natural process of repair.

Hört (9) has recently put forward a theory that ulceration is due to a deficiency in the patient's serum of bodies which normally prevent autolysis or self-destruction of tissue. He suggests that gastric ulcer is produced by the action of gastrolytic toxins and enzymes, and believes that one of the reasons why such an ulcer is difficult to heal is that its thickened floor prevents healthy lymph reaching it and preventing autolysis.

#### Pathological Anatomy.

Gastric ulcer is found in the parts of the digestive/

digestive tract which are exposed to the action of the gastric juice - namely, the stomach, the first part of the duodenum and the lowest part of the oesophagus, and in a few cases in the jejunum after gastro-jejunostomy.

Number. Only one ulcer is found as a rule, but occasionally two or more may occur. From the report of the Fenwicks on 867 cases of open ulcers it appears to be multiple in about 20% of all cases. Cases have been reported in which a large number of ulcers have been found as in that of Berthold - 34, and of Lange in whose case they were innumerable.

Situation. The most common seat of gastric ulcer is near the pylorus and in the majority of cases on the posterior wall near the lesser curvature. The anterior wall is less often affected and as a rule the ulcer is then acute. The greater curvature and the fundus are rare sites of ulceration. Only 20% of all ulcers are situated outside a circumscribed area of the stomach corresponding to the posterior wall, the lesser curvature and the pyloric portion.

Size. In size the ulcers vary greatly but generally are under one inch in diameter; but they are frequently smaller or they may be larger, e.g. Cruveilhier reported a case in which the ulcer measured  $6\frac{1}{2} \times 3\frac{1}{2}$  inches. Small ulcers are usually acute.

Appearance/

Appearance. The appearance of an ulcer varies according to whether it is acute and of recent origin or chronic. The acute ulcer is round or oval in shape and is characterised by having the appearance of having been punched out of the stomach wall: its edges are smooth, clean cut and show no thickening: the floor is, as a rule, smooth, but may be irregular, firm or soft. Small haemorrhages may occur in the edges or immediate neighbourhood of the ulcer. The depth of the ulcer varies according as to whether it has only involved the mucosa or extended deeper into the muscular or serous layers. As the ulcer deepens it tends to destroy a lesser area of each succeeding coat and thus it may have a funnel shape with sloping or terraced walls and frequently its axis is oblique following the line of a branch of the coronary artery.

In ulcers of some duration, i.e. in chronic ulcers, the appearances are altered by inflammatory changes taking place around the edges and base. Thus such an ulcer is usually larger and may be irregular in shape due to cicatrisation. The edges are thickened and the funnel shape of the acute ulcer is lost as the destruction of tissue at the base in the deeper layers may be as great or even greater than at the mucosa. The floor of the ulcer may be irregular and is firm, due to formation of fibrous tissue. Around the ulcer there is often a chronic inflammatory condition and the/

the peritoneum over the ulcer is usually thickened due to fibrinous deposits in it. This condition is frequently of service to surgeons in localising an ulcer. The chronic inflammatory thickening may be so great as to form a definite tumour and thus complicate the diagnosis of simple ulcer and malignant disease.

Histologically the sides and floor of an acute ulcer only show destructive changes without signs of inflammation which, however, is evident in chronic ulcers. Although some observers hold the opinion that inflammation is a constant and primary condition in the walls of the stomach in gastric ulcer, many pathologists regard it as secondary and variable.

If an ulcer penetrates deeply and inflammation of the serous coat over its base occurs, adhesions between the stomach and an adjacent organ or organs are formed. These adhesions, which are at first fibrinous and later fibrous, are most frequently met with between the stomach and pancreas and the left lobe of the liver and in this way the stomach becomes adherent to these organs. Adhesions may act as a safeguard to the patient by preventing the ulcer from perforating into the general peritoneal cavity by forming the floor of the ulcer after the serous coat is ruptured. The adjacent viscera which may become adherent to the stomach are in order of frequency, Pancreas, Liver, Pancreas and Liver, Colon, Liver and Colon, Spleen, etc. When the ulcer is/



is situated on the anterior wall of the stomach the formation of adhesions is rare due to the greater mobility of this part of the organ and also to the absence of fixed viscera in this region. This fact accounts for the greater frequency with which ulcers in this situation perforate. Due to the formation of extensive adhesions there may be a considerable matting of the stomach and adjacent organs and thus the size and mobility of the stomach may be interfered with.

Perforation. When the ulcer penetrates to the serous coat and if firm adhesions have not formed, perforation may occur. This is most apt to occur when the ulcer is situated on the anterior wall of the stomach. Perforation occurred in 28.1% of 187 cases collected by Musser (Osler (10)). The results of perforation vary according to the site of the ulcer and the size of the perforation. If perforation takes place into the general peritoneal cavity, as is generally the case with acute ulcers on the anterior wall, general peritonitis is set up. Ulcers situated on the posterior wall perforate into the lesser sac of peritoneum and may thus give rise to an air containing abscess. The ulcer may perforate into an adjacent hollow viscus to which the stomach is adherent. In this case a bimucous fistula is established. If the perforation is small, the leakage from the stomach is slight and may only give rise to localised peritonitis and/



and in this way the opening may be closed or it may be the origin of a localised abscess, e.g. a subphrenic or an abscess between the stomach and the anterior abdominal wall. Cases of perforation into the pleura, pericardium and heart have been recorded.

Erosion of blood vessels. Due to the destructive process by which a gastric ulcer progresses a blood vessel in its path may be eroded and give rise to haemorrhage. This is equally common to the acute and chronic varieties of ulcer. This accident may be prevented by the formation of a thrombus in the open vessel which frequently shows evidence of peri- and end-arteritis. The haemorrhage varies in quantity according to whether a small or large vessel is eroded. If the vessel is capillary the blood may not be visible to the naked eye in the vomit or faeces. Boas termed this "occult haemorrhage". Severe, or it may be even fatal haemorrhage is the result of erosion of a large vessel, e.g. the coronary artery or the splenic artery, or again it may arise from erosion of an adjacent vascular organ such as the spleen or liver.

Cicatrization. From the evidence obtained from autopsies, ulcer of the stomach naturally tends to heal by cicatrization as manifested by the scars which are so frequently found. Superficial ulcers frequently/

frequently heal and leave practically no scar or deformity. Deep ulcers on the other hand leave a well marked cicatrix which by its contraction may lead to one or other of the troublesome sequelae of ulcer of the stomach. The situation of a cicatrix determines the character of this. Thus a cicatrix situated at or near to the pylorus may lead to pyloric stenosis and this is the most frequent sequel of ulcer and leads to dilatation of the stomach. Again an ulcer situated on the lesser curvature and extending transversely to the long axis of the stomach by its contraction may lead to the "hour glass" or "bilocular" stomach in which the stomach is divided into two pouches communicating by an opening of varying size. The mucosa of the cardiac portion, which is generally the larger, is smooth and the wall is thin whereas the pyloric portion has a thickened mucosa and frequently shows signs of inflammation. An ulcer in this situation may also lead to a deformity in which the cardiac and pyloric openings are dragged together and bound by adhesions.

Perigastritis and adhesions may also cause deformity of the organ.

#### Symptoms.

In a great many instances the history of the case makes the diagnosis certain the symptoms alone being definite/

definite, or it may be difficult or altogether impossible, the symptoms being obscure or latent.

In most cases in addition to dyspeptic symptoms there are three manifestations of gastric ulcer which are regarded as characteristic of that disease, namely pain, vomiting and haematemesis.

Pain is probably the most prominent and constant symptom of ulcer of the stomach. It is pain characterised in several ways - it is paroxysmal: it is localised: it occurs during digestion and is influenced by the character of the ingestion<sup>a</sup>. Occasionally the pain is continuous with acute exacerbations after the ingestion of food. In some cases the pain comes on almost immediately after food is taken and may be due to the irritation of the ulcer by the food. Solid food as a rule causes pain more readily than liquid food. In other cases the pain is delayed for some time after food and in a few cases a few hours may intervene between the ingestion of food and the onset of pain. In these cases the movements of the stomach and the increased secretion of acid gastric juice and the passage of food through the pylorus may be the cause of the pain. It is as a rule situated in the epigastrium and is frequently localised to a small area which corresponds to that of tenderness on pressure. Occasionally the pain radiates either to right or left and in chronic cases it is situated at a lower/

lower level and is also more diffuse. In addition to epigastric pain it is not uncommon for patients to complain of a painful area in the back usually to the left of the spine at the level of the tenth dorsal to the first lumbar vertebrae or it may be situate over the spine at the level of the sixth to eighth dorsal vertebrae. The character and intensity of the pain vary very considerably in different cases. The patient may complain of a "sore spot" or simply of a diffuse sense of pressure, a sensation of heaviness in the epigastrium or it may be described as stabbing, boring, burning or gnawing in character. But the most characteristic form is that in which the pain comes on in paroxysms and is intensely severe (gastralgia). This pain lasts for a variable time and as a rule until the stomach is emptied either by vomiting or by the process of digestion. In a few cases the pain starts before food is taken. Remissions from pain for days or even weeks occur but during this time tenderness may be elicited on pressure. There is sometimes a continuous pain, dull and aching in character and more diffuse due to dyspepsia. In some cases pain is absent altogether.

The dorsal pain is a referred or somatic pain (Ross) and patients frequently complain of intercostal pain and also in the left shoulder. Singer and others regard the pain which radiates from the epigastrium/



epigastrium to the costal arches and along the inter-costal nerves to the spine as a valuable early diagnostic sign.

Corresponding to the areas of pain and tenderness hyperaesthetic areas of skin over them are to be made out. The area of hyperaesthesia in the epigastrium is strictly localised and is usually about half an inch in diameter situated most frequently between the ensiform and left costal margin (Drs. Head & Mackenzie). This was regarded as a valuable sign until recently when it was pointed out that it is frequently absent and also occurs in anaemia and neurasthenic dyspepsias. Patients sometimes obtain relief from the severe attacks of pain by adopting a certain posture. Thus in some cases relief is got by assuming a bent attitude in which the abdominal walls are relaxed; in others a horizontal, a prone, or a lateral position is adopted. Some authors have made use of this as a guide to the location of the ulcer on the assumption that the relief obtained is due to the contents falling away from and thus ceasing to irritate the ulcer. Others, however, regard the effect obtained as pointing to the presence of some complication such as adhesions, and Schmidt (11) points out that the same thing occurs in painful affections of other organs such as the liver, kidney, appendix, etc., and regards it rather as being due less to a displacement of the gastric/



gastric contents than to that of the stomach itself.

Many theories have been advanced as to the mechanism of pain in gastric ulcer, none of which are universally accepted.

Vomiting. While vomiting is an important symptom of gastric ulcer it is by no means a constant one. It may occur at any stage during the course of the disease and be due to various causes. Early in the disease vomiting immediately after food is probably due to irritability of the stomach. As a general rule it does not occur until two or three hours after the ingestion of food and then occurs at the height of a paroxysm of pain which it immediately relieves, and is then due to the contact and irritation of food on the ulcer. In many cases it appears to be influenced by the nature of the food. Thus liquid food causes vomiting less frequently than solid food.

The character of the vomit varies and its appearance may be of value in diagnosis. It consists of food either unaltered or partly digested according to whether it is immediate or delayed. The taste is acid and the vomit contains an excess of hydrochloric acid. "Bilious" vomiting rarely occurs in gastric ulcer.

Haematemesis or haemorrhage. This is one of the cardinal symptoms and occurs in about one third of all cases. It may be the first symptom of gastric ulcer/

ulcer and occur in patients who considered themselves quite healthy or who suffered only from a slight degree of indigestion. As a rule, however, it occurs after other symptoms are fully established. The amount of haemorrhage may be very slight and the blood may not be apparent to the naked eye in the vomit or in the faeces as in some cases it is not vomited but passes into the intestines. Thus special tests may be required for its detection and several such tests are available, such as the guaiac and the more delicate benzidin tests. Boas gave the name "occult" to such haemorrhages and drew special attention to their diagnostic and prognostic significance in doubtful cases. In other cases there is moderate or even profuse haemorrhage. In moderate haemorrhage the blood may not be vomited but passed per rectum as melaena and give to the stools a dark or tarry appearance. If the bleeding takes place slowly the blood is acted upon by the gastric juice and may be vomited as "coffee grounds" material. This change is due to the haemoglobin being converted into haematin. When haemorrhage is profuse the blood is vomited either liquid and unchanged or in the form of large clots.

As a rule, before a severe haematemesis the patient has premonitory symptoms - a "feeling as of something giving way": a sensation of distension: or it may be a sharp stabbing pain: the patient feels faint/

faint, complains of a peculiar taste in the mouth and nausea and then suddenly vomits quantities of blood without retching. This may be repeated several times for a few days. The general condition of the patient is that common to all forms of severe haemorrhage. Rarely is haemorrhage from an ulcer fatal and when it is, it is due to rupture of a large vessel and in these cases death may occur before any blood is vomited. Several attacks of severe haematemesis may cause death through exhaustion, but as a rule the patient gradually recovers and is in a very anaemic condition. Haemorrhage alone is not of much assistance in diagnosing gastric ulcer, as in various other diseases of the stomach, such as erosions and carcinoma and also in cirrhosis of the liver, etc., haematemesis is common, but in conjunction with a history of pain and vomiting especially in relation to meals it forms a very important link in the evidence in favour.

Besides these three cardinal symptoms of gastric ulcer there are others which it is convenient to group under the headings of the various symptoms:-

Gastro-Intestinal. As a rule the appetite is unimpaired and when it is it is through fear of causing a paroxysm of pain by eating. The condition of the tongue varies - it is usually clean but in cases accompanied by chlorosis it may be pale and flabby and in chronic cases may be covered by a white fur due to accompanying/

accompanying gastritis.

Constipation is very common and diarrhoea rarely occurs.

The majority of patients with gastric ulcer have either carious or artificial teeth, the latter showing that previously they had decayed teeth.

Circulatory. There is seldom any evidence of organic heart disease although haemic murmurs are common. There may be slight irregularity of the heart's action. Chlorosis is very common and the blood has the characteristics common to it. The red blood corpuscles are diminished and the count may be very low (two millions per c.mm.). Osler quotes a case in which the red blood corpuscles fell to 700,000 per c.mm.. The arteries very rarely show signs of degenerative changes.

Genito-urinary. In the majority of cases the urine is normal but its reaction may only be very slightly acid or even alkaline due to the hypersecretion of hydrochloric acid. Acetone and di-acetic acid are of comparative frequent occurrence in the urine of patients with diseases of the gastro-intestinal tract including gastric ulcer. Their presence may give rise to no symptoms or symptoms resembling the toxaemia of diabetic coma may develop. Lorenz has recorded five cases in which this developed and Professor Dreschfeld (12) reports one.

Albumin/



Albumin is occasionally present.

Except in cases due to anaemia the menstrual functions are normal.

The temperature is normal or subnormal. A slight rise of temperature is not uncommon after haematemesis and also occurs with complications and in intercurrent disease.

Nervous system. Headache, giddiness and sleeplessness are fairly frequent and are probably due to the anaemia. Patients with gastric ulcer may develop neurasthenia or may become melancholic or hysterical. A rare but very grave complication is tetany.

General health. This varies very much and depends a great deal upon the duration of the disease, the course it runs and the age of the patient. In young persons the nutrition remains good for some time and the appearance is only that of anaemia. In older patients and especially if the disease has lasted for some time with either severe pain or attacks of haematemesis and vomiting, signs of wasting are evident and they assume a pinched, pale and anxious expression - the "abdominal facies".

Physical examination of a patient suffering from gastric ulcer may reveal nothing beyond epigastric tenderness. In some chronic cases with marked emaciation it is said that a fibroid cicatrix may be felt. Along with the epigastric tenderness there are/

are corresponding areas of hypersensitive skin.

In cases in which inflammation has occurred from matting together by adhesions a tumour may frequently be palpated and a diagnosis of malignant disease made which is only falsified by its duration or in some cases by its total disappearance after gastroenterostomy.

Dilatation of the stomach can be made out in cases with pyloric obstruction.

#### Complications and Sequelae.

These are numerous and varied in gastric ulcer. Perforation, general and localised peritonitis, perigastric adhesions, abscess formation and stenosis are the more important.

Perforation is common and of all the complications is the most serious and dangerous to life. It is commoner in women than in men. The results of perforation depend to a certain extent upon its size and situation. Acute ulcers are more prone to perforate than the chronic. Perforation frequently causes death by peritonitis. In a few rare cases a small perforation has been known to be closed without any untoward result. In other cases it may lead to the formation of a localised abscess or to a fistula between the stomach and another hollow viscus. Perforation into the general peritoneal cavity occurs in about 6.5 per cent of all cases of gastric ulcer (Welch)./

(Welch). An ulcer situated on the anterior wall of the stomach is much more frequently the cause of this than one situated on the posterior wall.

Perforation may occur as the primary indication of ulcer but more frequently it takes place after symptoms have been manifest for some time.

The occurrence of perforation is associated with characteristic symptoms. The patient experiences agonising pain either continuous or colicky in character in the epigastrium and later all over the abdomen. The patient is collapsed with a thin, thready and rapid pulse, the extremities are cold and a cold perspiration breaks out all over him; the face is pale and pinched, the eyes sunken and the expression is one of anxiety (*facies hippocratica*), the breathing is rapid and shallow and the voice is weak: the temperature is subnormal: the tongue is parched and the patient complains of thirst: there may or may not be vomiting. On physical examination of the abdomen movement is absent or diminished and there is distension. On palpation there is marked tenderness in the epigastrium and the muscles are on guard. On percussion the note is tympanitic and the liver dulness is either diminished or lost. The patient may die at this stage from collapse, but if he survives this period it is followed by one which is deceptive and in which the pain is less and the patient more comfortable in/

in every way. But in a few hours signs of peritonitis set in and unless the patient is speedily transferred to the care of a surgeon death results in the great majority of cases.

Perigastric adhesions, if formed in the neighbourhood of the pylorus, may produce a kinking of the duodenum with secondary dilatation. Various other deformities of the stomach may be due to adhesions and also its functions may be interfered with.

Pain is the chief symptom and frequently follows the taking of food and is also influenced by walking and certain movements, e.g. stretching. It is dragging in character and is relieved by the assumption of the horizontal position.

Abscess formation results when perforation takes place and gives rise to localised peritonitis, the escaped stomach contents being shut off from the general peritoneal cavity by previously formed adhesions. This frequently occurs when the perforating ulcer is situated on the posterior wall of the stomach. At first the symptoms are those of general peritonitis but these subside and the patient is more comfortable. But his condition remains unsatisfactory - the pulse is rapid, the temperature swings up and down and there are occasional rigors and profuse night sweats, and in the long run the patient may die from exhaustion. In other cases the abscess may burst into the general peritoneal/



peritoneal cavity and then causes death by peritonitis: in rare cases it may perforate the diaphragm. In some cases operative measures are possible and thus the patient is saved. Stenosis of the pylorus due to the contraction of a cicatrix situated upon it or in its neighbourhood causes dilatation of the stomach with its symptoms.

Carcinoma of the stomach. Many observers regard chronic gastric ulcer as a predisposing cause of cancer and frequently cases have been reported in which the cancerous infiltration has commenced around the edge or in the cicatrix of an ulcer.

Subcutaneous emphysema following perforation is a very rare complication.

#### Duration and Course.

Anatomically and clinically ulcers have been divided into two classes - acute and chronic - but there can be no hard and fast line between these. The length of time an ulcer has existed is a doubtful question as so many ulcers remain latent until revealed either by haematemesis or perforation. Many ulcers undoubtedly go on for years, the patients having intervals of relief from symptoms. Again the course of an ulcer may be very rapid, terminating quickly in perforation or haematemesis.

As/

As the majority of ulcers are amenable to treatment, their course and duration will depend greatly upon how soon after it first becomes manifest appropriate measures of prevention and cure are taken.

### Diagnosis.

The diagnosis of gastric ulcer may be easy or it may be very difficult or even impossible, according to whether the history be definite or indefinite and the symptoms are present or absent. No symptom alone is characteristic of gastric ulcer and all may be due to causes other than ulceration. To complete the diagnosis it may be necessary to keep the patient under observation for some time.

A history of pain in the epigastrium following the taking of food, vomiting some time afterwards which relieves the pain, and haematemesis is the most reliable combination of symptoms, and a long localised epigastric tenderness and hyperacidity of the vomitus, the most characteristic.

Haemorrhage from a gastric ulcer is to be distinguished from haemorrhage from some source outside the stomach, the blood having been swallowed and then vomited. In such cases the haematemesis is generally slight and frequent. Haematemesis may also occur in the course of any of the "bleeding diseases". It occurs also, in cardiac disease and cirrhosis of the liver, /

liver, as a result of ruptured varicose veins "oesophageal varices". Numerous superficial erosions of the mucous membrane may be the origin of severe haemorrhage and Hale White has suggested that haematemesis in many anaemic females, generally attributed to gastric ulcer or to vicarious menstruation, is an entirely independant disease to which he has applied the term "gastrostaxis".

Gastric catarrh and Dyspepsia. Acute gastric catarrh gives a quite different clinical picture from gastric ulcer. It is the subacute and chronic types which may be confounded with ulcer. In these there is diffuse pain frequently relieved by pressure and never so severe: flatulence and discomfort are prominent symptoms and last for a long time after meals: vomiting varies very much in time and the gastric juice is not abnormally acid: haematemesis is very rare.

Hyperchlorhydria although giving rise to symptoms similar to those of gastric ulcer, does not cause haematemesis, the pain is diffuse and pressure may give relief from it or there may be no tenderness. In gastralgia the pain is as equally severe after fluids as after solids or has no relationship to the taking of food: pressure frequently relieves the pain: vomiting is not constant and haematemesis does not occur.

Gastric crises. In these the attacks of pain and vomiting/

vomiting very often have no relation to food. Haematemesis rarely occurs. The unmistakable signs of Locomotor Ataxia are present.

Carcinoma occurs most frequently after middle life.

The pain is continuous and present even when the stomach is empty. The appetite is impaired. Vomiting is a later manifestation and haematemesis is usually slight or "coffee grounds" in character. A tumour may be palpable. Examination of the stomach contents shows a diminished secretion or absence of hydrochloric acid and lactic acid and the Boas-Oppler Bacillus are present. Wasting is more marked and the patient is cachectic.

Gall-stones. The pain of gall-stone colic is sudden in onset and frequently occurs some hours after unsuitable diet. The pain is excruciating and colicky in character and is referred to the right hypochondrium as a rule: there is a referred pain in the right shoulder. Tenderness on pressure quickly disappears after the attack of colic is over. Vomiting may usher in the attack and continue throughout it. The vomit is "bilious" in character. The temperature is raised and the conjunctivae may be coloured.

Movable kidney although associated with attacks of abdominal pain and vomiting does not greatly inconvenience the diagnostician. The situation and character of the pain, the absence of epigastric tenderness and/



and haematemesis and in the majority of cases a neurasthenic condition help to distinguish this complaint from gastric ulcer.

### Prognosis.

In the vast majority of cases a favourable prognosis can be given, especially if the patient is seen at an early date and appropriate treatment commenced.

Relapses are common and in complicated cases relief may only be obtainable by surgical treatment.

In the majority of cases of gastric ulcer, a fully extended course of medical treatment should be given before resorting to surgical measures except in those cases presenting special features.

Danger to life from gastric ulcer may arise from perforation and general peritonitis or from haemorrhage.

### Various Modes of Treatment.

In the majority of cases gastric ulcer is curable, as has been demonstrated by observation at post-mortem examinations. Thus in the treatment of gastric ulcer our aim should be to promote healing and the cicatrization of the ulcerated surface. To do this it is of the first importance to allay all irritation, both mechanical and functional. Just as in the treatment of/

of a wound of an external surface we put the part at rest, so in the treatment of a wound of the gastric wall in the form of an ulcer, rest is of paramount importance. Therefore all modes of treatment which are adopted with the object of allowing the patient to follow his usual habits are unsatisfactory, if not futile. The peculiar conditions which are present in the stomach and which tend to prolong the treatment (unduly to the lay mind) are:- the peristaltic contractions of the stomach wall: the hyperacidity of the gastric juice and the mechanical irritation of food. In our treatment we must adopt methods which will overcome these retarding influences to the healing process and thereby produce the best possible conditions for the complete cure of the lesion.

Rest has been for long recognised both in this country and on the Continent as the basis of all treatment. The patient must be kept in bed, not even getting up to micturate or defaecate. The stomach must be kept as near as possible in a quiescent state by keeping it as empty as possible, thus avoiding stretching of the ulcerated surface and allowing the mucous membrane to fall into folds. Also mechanical irritation by food is diminished and the secretion of hyperacid juice is lessened. The most important point in the treatment comes to be one of nourishing the patient in a way which gives the stomach the least amount/

amount of work to do. Medicinal treatment may be of value, especially in regard to the relief of symptoms.

Most authorities start treatment by giving the stomach complete rest by stopping all food by the mouth and nourishing their patients by rectal alimentation by giving nutrient enemata. Dr. Donkin (13) was the first to adopt this procedure and reported excellent results in a large number of cases of gastric ulcer. But it has been found that this method is not altogether satisfactory and that there is danger of inanition due to the poor absorptive powers of the rectal mucous membrane. Also certain unpleasant complications may arise such as parotitis, and many physicians therefore reserve this method of treatment for cases complicated by haematemesis or in which any food by the mouth gives rise to severe pain or intractable vomiting. Most physicians have their own receipt for a nutrient enema and many and various articles of diet are used, e.g. raw eggs, beef tea, brandy, predigested food, etc., etc., and many elaborate nutrient enemata are recommended. The best of these all contain either dextrose, salt, powdered peptone or casein. In view of Pasteur's results with warm water injections it is extremely doubtful if it is necessary to load the rectum with articles of diet. To supplement rectal feeding subcutaneous injections of various sterilized preparations such as olive oil and/

and dextrose solutions have been recommended.

After the preliminary stage of rectal alimentation, feeding by the mouth is gradually instituted.

Milk has always held the premier position in the dietary considered suitable in cases of gastric ulcer and much can be said for it as an appropriate food. It passes quickly from the stomach without causing much peristaltic action: it decreases hyperacidity because of its alkalinity: it is non-irritant. It should be given in small quantities, i.e. from four to six ounces at a time and is frequently diluted as this prevents curdling. The favourite diluents are lime water, soda water and bicarbonate of soda, and these also enhance its antacid properties, or it may be given peptonized. To some patients milk is intolerable and substitutes such as butter milk, milk deprived of some of its casein or clotted with rennet are employed. But in some cases milk in any form is nauseous and other articles of diet such as beef tea, meat extracts or jellies are preferred and Dreschfeld strongly recommends scraped raw beef (14).

An exclusively milk diet requires to be supplemented comparatively early in the course of treatment, about the tenth day, and proteid in the form of white of egg and fat, either cream or butter, and food such as Bengers along with glucose and arrowroot may be added.

This supplemented diet is continued for a week or two/



two and after this the patient is gradually worked up through a soft farinaceous diet until ultimately when there is every likelihood that cicatrisation is complete and all symptoms and signs have been negative for some time more latitude in the matter of food is permitted. For several months after this, discretion in the choice of articles of diet has to be exercised. Alcoholic beverages should not be allowed. Lavage of the stomach has been advocated by some authorities but is condemned by others owing to the degrees of traumatism and haemorrhage.

A method of procedure which avoids the dangers of practicing lavage has been recommended (15). It consists in washing out the alimentary tract by giving repeated draughts of Carlsbad water or salts in solution in warm water. This is carried out as follows:- "A teaspoonful of Carlsbad salts is dissolved in six ounces of warm water and four such doses are given at intervals of ten or fifteen minutes in the morning fasting. Breakfast must not be taken until half an hour after the last dose" (Burney Yeo). The result of this is that after breakfast the bowels are evacuated two or three times, the stools being loose, the stomach is cleansed of mucous and the salts have also an antacid action.

In Germany the method of treating gastric ulcer by what is known as the "rest cure" is well established/

established. The "cure" requires the patient to have absolute rest in bed for two or three weeks. During the first week the patient is fed exclusively by the rectum for several days (Riegel); warm compresses are applied to the abdomen and thirst is relieved by allowing small fragments of ice to be sucked and by frequently rinsing out the mouth. From two to four nutritive enemata are given pro die. After six or eight days or even longer small quantities of food are given by the mouth. As a rule the diet consists of milk, given at first in very small quantities and diluted with barley, or lime, or soda water. To this bouillon or meat jelly or Leube's meat solution is frequently added as milk itself cannot sustain the patient. Raw, beaten white of egg and dextrose are also allowed. After some time, but not before the tenth day and if the condition of the patient is favourable, the second dietary is begun. This consists of gruel, tapioca pudding, boiled chicken or pigeon or calves' brains or thymus. Finally scraped raw beef is also included. Again, after a further interval and circumstances being favourable, the third dietary is given consisting of beef steak, scraped or broiled, scraped raw ham, potatoes, cauliflower and zwieback. As late in the treatment as possible the fourth dietary is instituted and consists of fowl, pigeon, roast beef, venison, veal, meat and flour soups, trout/

trout, pike or shad and souffles.

The medicinal part of the treatment consists in giving alkaline, saline mineral waters or solutions of their salts. Carlsbad water or salts is the favourite. After a fortnight, if the patient is free from pain he is allowed to sit up for a short time each day and this liberty is gradually increased. In uncomplicated cases the cure covers a period of from four to six weeks and after this for several months certain restrictions in diet are necessary.

Lenhartz in recent years has introduced a method of treating gastric ulcer consisting of a concentrated egg albumin diet with the aim of (a) promoting healing by giving as much nourishing food as possible; (b) preventing interference with healing by the food containing a large quantity of proteid and thus "fixing" the excessive acid secretion; (c) preventing distension of the stomach.

The essentials of the treatment are as follows (16). Absolute rest in bed for four weeks. During the first fortnight an ice bag is applied continuously to the abdomen. The object of the ice bag is to prevent gaseous distension of the stomach, to relieve pain and to favour contraction of the stomach. On the first day seven to ten ounces of iced milk is given in spoonfuls along with two to four beaten up eggs. The eggs/

eggs are beaten up with a little sugar and a little wine may be added. This is given even if haemorrhage has been recent. At the same time Bismuth Subnitrate is given in thirty grain doses three times a day for ten days. On each succeeding day an addition is made to the diet of  $3\frac{1}{2}$  ounces of milk and one egg. Thus at the end of the first week the patient gets one and a half pints of milk and six or eight eggs in twenty-four hours. These quantities are continued for another week. About the sixth day 35 grammes of raw minced meat is added to the diet and if this is well borne the quantity is doubled next day and may be further increased later. It is either given alone or mixed up with the eggs. The patient is now allowed well cooked rice and a few softened zwieback. In the third week an advance is made to a light mixed diet with strict injunctions to masticate thoroughly.

The bowels are not evacuated during the first week and only in the second week by small injections of warm water or glycerine. After this an enema is given daily if they cannot be regulated by means of food.

Blaud's pills softened with glycerine are given thrice daily at the end of the first week.

Senator has modified the Lenhartz treatment. He gives a diet including gelatin fat and sugar. "Gelatin is easily digested, possesses a high nutritive value/



value being a saver of proteid and it increases the coagulability of the blood and thereby helps to stop bleeding" (Dreschfeld).

He uses gelatin in cases of haematemesis. He also gives fresh butter and cream and thus gives a diet of higher calorific value than Lenhartz's.

Although reports in favour of Lenhartz treatment are numerous there is the obvious objection to it, which is supported by many eminent authorities, of giving food by the mouth immediately after a haematemesis. This practically does away with all the advantages of Lenhartz treatment in the early stages of gastric ulcer.

Hörtl (17) recommends the use of normal horse serum by oral administration and reports good results from this treatment. He gives a minimum of thirty cubic centimetres in the twenty-four hours and in cases with severe pain or continued haemorrhage he gives up to sixty or eighty cubic centimetres per diem. The serum is given in half an ounce of cold water and it must be fresh, atoxic and sterile. In severe cases he continues its administration for six weeks.

#### Medicinal Treatment.

By itself medicinal treatment of gastric ulcer is useless. As an adjuvant to any rational mode of treatment it may hold a place, but many physicians regard it as unnecessary and at the best only supplementary/

supplementary. However, many medicines are in use and may be considered under three headings as

- (1) having a direct action on the ulcer itself, or
- (2) giving relief to certain symptoms, and (3) for the arrest of haemorrhage.

Bismuth, either in the form of carbonate or subnitrate has long been regarded as the efficacious drug. It is given in powder or suspension in ten to twenty grain doses and is supposed to act chiefly by forming a protective pellicle over the ulcer and thus favouring cicatrisation. Bismuth salts are frequently given along with alkaline carbonates - sodium bicarbonate and magnesium carbonate and this combination along with a milk diet is probably the most popular method of treating gastric ulcer. On the Continent much larger doses of bismuth are given than is the practice in this country. Fleiner and others adopt the practice of giving it in suspension in water by means of a stomach tube. The patient then assumes the posture which will bring the bismuth in contact with the ulcerated surface, and after fifteen minutes it is drawn off again. An insuperable objection to this mode of treatment lies in the use of the stomach tube with the risks attached.

Olive oil in two to four ounce doses given by means of the stomach tube after a preliminary wash out has been recommended.

Another very old remedy is Nitrate of Silver, and/

and is strongly recommended by many. It is given either in the form of a pill (argenti nitrat<sup>is</sup> gr. $\frac{1}{3}$ ) three or four times a day before food or in solution.

Drugs used for the relief of symptoms - pain and vomiting include the foregoing as these have been found to exert sedative properties. In addition to these and such external remedies as the application of ice, hot fomentations and counter irritants, we have many others to fall back upon. Thus the occasional use of opium in chronic cases may be necessary to relieve severe attacks of pain and vomiting. Morphine in addition to its sedative qualities has, according to Professor Schäfer and others, a beneficial action in that it diminishes the secretion of hydrochloric acid when given subcutaneously. Riegel, on the other hand, holds that the exact opposite follows its administration. Dilute hydrocyanic acid (3-5 minims), creosote ( $\frac{1}{2}$  - 1 minim) and cocaine hydrochloride ( $\frac{1}{3}$  gr.) are also useful.

#### Treatment of Haematemesis.

Absolute rest in bed is essential with the head low. All food by the mouth is stopped and rectal feeding substituted. The patient should be reassured as to his condition and in addition to its sedative action generally, a hypodermic injection one third of a grain of morphine, will greatly help in soothing the nervous and agitated patient.

Thirst/

Thirst is relieved by allowing small fragments of ice to be sucked.

If haemorrhage continues or recurs, it will be necessary to employ further measures. Any drug which raises the vascular pressure is to be avoided. The most powerful local styptic is probably active suprenal extract. Adrenalin chloride in solution of a strength of 1 in 1000 is a most suitable preparation in cases of gastric ulcer (18). When given by the mouth it has been found that it does not raise the blood pressure. It is active when diluted and it does not destroy the tissues and thus interfere with the healing process. It should be given at short intervals of not more than an hour as after its action is over it causes vaso-dilatation. Grünbaum recommends that it should be given in drachm doses along with one grain of neutral calcium chloride freely diluted. If haemorrhage still continues other styptics may be tried, and of these turpentine given in capsules or as an emulsion is probably the most satisfactory. Ferric perchloride, lead acetate, tannin, alum may under certain circumstances be useful.

The intramuscular injection of gelatine has been recommended.

If haemorrhage is so severe as to endanger the life of the patient, subcutaneous injections of normal saline may be necessary. A pint of saline solution is/



is allowed to flow slowly through tubing connected with a funnel into the loose subcutaneous tissues of the axillae or inner sides of the thigh.

### Surgical Treatment.

In all cases of gastric ulcer (excepting perforation) medical treatment should be given a thorough trial before surgical procedure is resorted to. It is only in cases in which complications are present and those which do not yield to medical measures which require surgical interference.

Musser Kelly (19) says:- "It is becoming more and more recognised that surgical treatment is applicable almost entirely to the complications and sequels of peptic ulcer. Surgery is applicable to the uncomplicated disease in rare and carefully selected cases only".

Operation may be necessary in the few cases of severe repeated haemorrhages or in those in which it resists all medical measures. If an ulcer is found, the general procedure is excision of the ulcer as affording the best result.

Perforation. The treatment of perforation is essentially surgical and its success depends very largely upon an early diagnosis and immediate operation. The treatment of perforation consists in the closing of the rupture in the stomach either by invagination of the ulcer or its excision. The death rate in cases of/

of perforation has been greatly reduced by early operative measures. Surgical treatment of gastric ulcer applies principally to these chronic cases which resist all medical treatment and to cases in which the general health of the patient is interfered with by reason of complications and sequelae interfering with the gastric functions. Thus operative measures may be indicated in chronic indurated ulcer, pyloric stenosis with dilatation, "hour glass stomach", in cases with adhesions or for subphrenic abscess, and also for the relief of intractable pain and vomiting. In the majority of these cases gastro-enterostomy is the operation of choice.

As a rule acute gastric ulcer is a medical disease and the large majority of cases are curable by medical means alone. "Failure in the medicinal treatment of uncomplicated cases of gastric or duodenal ulcer is usually attributable to faulty management" (Musser Kelly). In chronic cases it is frequently difficult to decide when medical treatment should be stopped and benefit be sought in surgical measures.

The after treatment of cases of cured gastric ulcer is governed by the condition of the patient.

Anaemia should be treated with mild preparations of iron. Constipation should be carefully guarded against. A dose of Carlsbad salts first thing in the morning or a course of some aperient mineral iron water/

water may be desirable. The patient should live a healthy, hygienic life and, if a female, should take special precautions at the menstrual period. In all cases when a diagnosis cannot be definitely established it is sound to treat the case as one of gastric ulcer because symptoms may be latent, and it is these cases which are apt to give rise to <sup>great anxiety by sudden perforation or</sup> profuse haematemesis. In any case even if an ulcer is not present, the strict dietetic treatment and rest can have no other effect than a beneficial one.

#### Particular Treatment.

The following method of treating cases of ulcer of the stomach is that to which I wish to call special attention. This method appears to me to be more rational and to carry out to a greater degree than any method of treatment, which I have previously mentioned, that "great and paramount indication of treatment of gastric ulcer", viz., to secure the greatest amount of rest possible for the stomach and thus promote healing and cicatrisation.

The treatment is based upon the proteid sparing properties of gelatin and although some authors (notably Senator) have used gelatin in the treatment of gastric ulcer, yet I believe the following mode of procedure to be original, having failed to find any account/

account of it in the literature on the subject.

I am indebted to Dr. Edward Turton, of Hull, for allowing me to use the notes on his cases and to write up the treatment as he originally formulated it in 1909. He has since introduced it into his private as well as hospital practice, and has had every reason to be satisfied with his results.

The following is an account of the treatment:-  
The patient is kept in bed for a period of not less than four weeks. The average in my series of cases was six weeks, the longest period covering sixty days and the shortest twenty-eight days. During this time he is not allowed to get up even to micturate or defaecate, and all exertion is avoided. The rest is both physical and mental.

In ordinary cases without haemorrhage, the regular routine treatment is begun at once. The patient is allowed in the ordinary sense of the word no food by the mouth, but is given two ounces of a ten per cent solution of gelatin every two hours per oram and at the same time rectal injections of saline solution every four hours. The injections are given with the patient lying on his left side and eight ounces of solution are allowed to flow very slowly through a large catheter into the rectum. Each injection should take twenty minutes or longer to give. The rectal injections and administration of gelatin are/



are continued for a week or ten days. At the end of the first week, if all the symptoms have gone, one pint of beef tea and one egg are allowed per oram. The beef tea is given in quantities of five ounces at a time and the egg is beaten up and given along with one of the portions of beef tea. At the end of ten days or so the injections of saline solution are stopped and another pint of beef tea and another egg added to the diet. This along with the gelatine is continued for another period of from four to seven days. Then the administration of gelatin is stopped and lightly cooked mince and potato are added to the diet. The mince is given in the quantity of four ounces along with two ounces mashed potato and this forms the mid-day meal. Thus during the third week the patient gets in twenty-four hours two pints of beef tea, two eggs, four ounces of mince and two ounces of potato. In about another week, if the patient tolerates this diet and his condition is satisfactory, the quantity of mince is increased to a full helping. After this, at intervals of about a week he is gradually worked up through minced, fancy or ordinary diet until finally about the end of the fourth or fifth week he is allowed full ordinary diet.

In cases in which haematemesis has occurred the treatment requires practically no modification. The gelatin and rectal injections of saline are given as before/



before but are continued for a longer period without addition being made in the form of food. Besides the dietetic management of the cases there are other very important points of treatment.

All sources of septic infection must be eliminated. The removal of all carious teeth is very important and all oral sepsis prevented. This should be done as early as possible. The buccal cavity is kept sweet by frequently rinsing it out with a mouth wash of hydrogen peroxide.

Constipation is prevented by a daily wash out of the lower bowel and later by giving Mistura Alba, one ounce t.i.d. after food.

Anaemia is treated with a mild preparation of iron such as ferri et Ammon. Cit. gr. X, t.i.d. after the fourth week. Thirst is relieved by rinsing out the mouth.

Pain, if it does not yield to gelatin, may be relieved by giving 20 c.c. of normal horse serum by the mouth. Convalescence is greatly accelerated and the patient's general condition greatly improved, especially those who are anaemic, by open air treatment after the rectal injections are stopped. Under these circumstances any loss of weight is rapidly regained.

This method of treatment is essentially a "rest cure" and is based upon the use of gelatin as a "proteid-sparer" and of rectal alimentation in the form/

form of saline injections. In this way the most favourable conditions are secured in the stomach for the healing and cicatrisation of the ulcer and at the same time the strength and general condition of the patient are sustained.

It is recognised that the best way to secure rest for the stomach is to stop all food by the mouth and substitute rectal alimentation. Although many physicians adopt this procedure as a preliminary stage in their treatment of gastric ulcer, the majority reserve it for those cases only in which haemorrhage has occurred. This attitude is probably due to the belief that it is impossible to maintain the patient in a satisfactory condition by rectal alimentation. Even in those cases where it is employed, the rectal feeding is continued for an inadequately short period or nutrient enemata are used with the idea that otherwise the patient rapidly loses flesh and consequently develops a condition of health unfavourable to the healing process. I am of the opinion that rectal alimentation can be carried out over a period up to three weeks or more without any serious loss of weight or inconvenience to the patient, and is quite compatible with a condition which favours healing and this without resorting to the use of nutrient enemata - a procedure of doubtful value.

Most of the work on rectal alimentation has been done/

done by Continental authorities and even among them there is a division of opinion as to the value of nutrient enemata. This is due to the fact that the absorptive power of the mucous membrane of the rectum is limited, and that only a very few of the ingredients of the so called nutritive enemata are absorbed by it. Many authors indeed hold the opinion that their value depends almost entirely upon the quantity of water and salts that they contain. Thus we have Pasteur (20) advocating the use of plain water or normal saline injections and this method has also been recommended by Sharkey (21) from his experience of it. They are both of the opinion that "rectal feeding" is unnecessary; that the general condition of the patient remains good; that healing is accelerated; that the patient is more comfortable and that nursing is simplified. In the method of treatment I have described this method was followed and I can endorse all that they claim for it. In every case it was found to be eminently satisfactory. It has many advantages over the method of giving nutrient enemata apart from the doubt regarding their usefulness due to non-absorption. Thus thirst is not so distressing, the mouth remaining moister and free from any unpleasant taste: craving for food is practically nil: rectal intolerance rarely occurs and the patient is cleaner and more comfortable in every way. In addition/



addition it is known that an increased outpouring of hydrochloric acid into the stomach follows the administration of a nutrient enema and this is a most undesirable feature in the treatment of gastric ulcer as hyperchlorhydria is regarded as a factor preventing spontaneous healing of the ulcer. This is not known to occur after the giving of a plain water or saline injection. Thus by the administration of saline injections into the rectum we get the stomach in a state of rest. But there was one great draw back to this method, or in fact to any method of rectal alimentation, and that was that loss of weight was inevitable.

In order to prevent this loss of weight and also to compensate, as far as possible, the want of nourishment the administration of gelatin by the mouth was adopted.

Gelatin, although it is similar to proteid in its elementary composition and in being converted into peptones by its digestion and also in its oxidation products in the body (urea,  $\text{CO}_2$ , and  $\text{H}_2\text{O}$ ), is unable to build up tissues and therefore cannot take the place of proteid as a food. But gelatin, owing to the rapidity and ease with which it undergoes decomposition in the body, diminishes the metabolism of proteid and in this way it becomes to a certain extent a substitute for proteid, that is it is a proteid/

proteid sparer. It is in this capacity that it is a most useful adjuvant in the rest cure of gastric ulcer.

As a proteid sparer gelatin can be classed along with carbohydrates and fats which also act in this way, but it is much more powerful than either of these. Voit has been the principal investigator of this subject and Schäfer (22) quotes an experiment of his demonstrating the superiority of gelatin as a sparer of proteid over fats or carbohydrates - a dog weighing thirty-two kilos was kept in a condition of nitrogenous equilibrium on a daily allowance of five hundred grammes of meat. When four hundred grammes of meat and two hundred grammes of gelatin were given, there was a gain of nitrogen to the body representing an increase of forty-four grammes of flesh, but when one hundred grammes of meat were replaced by two hundred grammes of fat or two hundred and fifty grammes of carbohydrates there was a loss of nitrogen to the body representing a loss of flesh equal to fifty and thirty-nine grammes respectively. Gelatin is the most powerful proteid sparer known. "By the administration of gelatin very large quantities of albumen can be spared in the body or devoted to increase of bulk" (Bauer (23)). According to Voit the amount of tissue proteid lost from the body can be reduced to about half of that normally lost. Thus although gelatin is not a "plastic" food, it is of great value in preventing/

preventing destruction of proteid.

In addition to its proteid sparing properties, gelatin has other characteristics which make its administration most advantageous in the treatment of gastric ulcer. Thus the digestion of gelatin is very easily accomplished. Hutchison (24) says that in this respect it is hardly surpassed by any food and quotes the case of a boy with a gastric fistula in whom complete peptonisation took place within an hour. Thus the amount of work required of the stomach for its digestion is practically negligible.

Gelatin also acts beneficially by fixing a considerable amount of acid during digestion, thus preventing irritation of the ulcerated surface and promoting healing. Schiff has classed it among the "peptogenic" substances, i.e. it promotes the secretion of gastric juice. Thus it will favour the more rapid and easier digestion of the food which is given in the early part of the treatment.

In cases of gastric ulcer complicated by haemorrhage there is no objection to the early administration of gelatin by the mouth because of its haemostatic properties. It acts as a styptic by increasing the coagulability of the blood. It has been recommended by several authorities in the form of subcutaneous injections for a considerable variety of haemorrhage. Michaelis (25) has used a 5% solution in cases of intestinal/

intestinal haemorrhage successfully, and Grace (26) has also found this method very effective. The same holds good also for oral administration. Whitla (27) says that in all probability gelatin given by the mouth acts as a local haemostatic in all forms of haemorrhage from the stomach. Manna (28), using it in this way, reports that he has obtained uniformly good results in all cases of intestinal haemorrhage.

Gelatin also has a certain influence in relieving pain. L. J. Facio(29) has found that, given subcutaneously, gelatin promptly relieves pain due to various causes. Certainly in pain due to gastric ulcer the relief obtained after the administration of gelatin is most marked.

I submit a series of twenty-nine cases treated by the foregoing method with, I hope to show, most encouraging results. These cases were collected from the records of the Hull Royal Infirmary and I personally was responsible for the management of seventeen of them.

Although the weight of the patients was obtained in a considerable number of cases, this was done with a view to ascertaining how much weight was lost whilst the patients were on gelatin and rectal saline and I certainly do not intend it to be regarded as a desirable or necessary part of the treatment.



C A S E S.

Case 1. C.E. Aet. 20. Female. Domestic.

Treated from May 29th to July 23rd. She gave a history of having attacks of pain in the epigastrium for six months. The pain came on immediately after taking food and was relieved by vomiting which occurred later. There was also referred pain in the back. No haematemesis and no meloena. She was slightly anaemic; the tongue was dirty but teeth were fairly good. There was localised epigastric tenderness in the mid line.

Treatment. Absolute rest in bed.

May 29th. Rectal injections every four hours of 3vi of saline solution and also 3 ii of a 10% solution of gelatin every two hours per oram.

June 4th. The above continued and 1 pint beef tea and 1 egg given by the mouth.

June 8th. Rectal injections stopped and another pint of beef tea and another egg added to the diet.

June 15th. Gelatin stopped and 4 oz. mince and 2 oz. potato added to the diet. Complained of slight pain after taking mince therefore return to diet of June 8th.

June 25th. Mince and potato again given and, being well borne, the amount of mince was increased on June 28th to 6 oz.

July 13th. Given fancy diet.

July 23rd. Discharged cured. On Balcony from July 2nd onwards. Weight on admission 6 st. 13 lbs. and this/

this was stated to be increased on discharge.

Complete rest covered six weeks.

Unable to be traced in December, 1913.

Case 2. A. S. Aet. 17. Female. Factory worker.

Treated from January 22nd to March 26th, 1909.

Gave a history of four months duration of epigastric pain coming on from 15 - 30 minutes after taking food.

Pain relieved by vomiting. No haematemesis and no meloena. During the last three weeks pain has been more severe and frequently vomited even after taking only milk. Always constipated. Development poor. Pale and anaemic looking. Teeth careous. Localised but slight tenderness in epigastrium.

Treatment. Absolute rest in bed.

January 22nd. Rectal injections of 3 vi of saline solution every four hours and 3ii 10% gelatin solution every two hours per oram.

January 26th. Allowed 1 pint beef tea and one egg by the mouth and taken off salines. Gelatin continued.

February 2nd. A further pint of beef tea and another egg given and gelatin continued. Also given Mist. Alba 3i t.i.d.

February 9th. Gelatin discontinued and 3 oz. mince and 2 oz. potato and thin bread and butter added to diet.

February 15th. Fancy diet given - all meat being minced, and iron tonic (mild) given t.i.d.

March 7th. Put on ordinary diet. Discharged March 26th - cured.

Weight on admission = 5 st.  $\frac{1}{2}$  lb. Whilst on gelatin and rectal salines weight remained constant and when discharged/

discharged = 6 st.

Could not be traced in November 1912.

Case 3. E. P. Aet. 28. Female. Housewife.

Treated August 23rd to September 24th 1909.

Gave a history of one year of repeated attacks of epigastric pain coming on immediately after meals and relieved by vomiting. Six weeks ago had a haematemesis and vomited "1 quart" of blood and since then, in spite of treatment, the pain and vomiting has been constant after food. A few days before admission she had a second attack of haematemesis. Has always been constipated. Very anaemic. Tongue furred. Tenderness in epigastrium and slight rigidity of recti especially over left rectus.

Treatment. Absolute rest in bed.

August 23rd. Rectal injections of  $\frac{3}{4}$ vi of saline solution every four hours and  $\frac{3}{4}$ ii 10% gelatin solution every two hours per oram.

August 27th. Saline given every six hours. Has now no pain and rigidity of recti has passed off.

August 29th. Injections discontinued and 1 pint beef tea and 2 eggs given per oram. Gelatin continued.

August 31st. Another pint of beef tea added to diet.

September 4th. Gelatin stopped and 4 oz. mince and 2 oz. potato added along with 2 pints peptonised milk.

September 14th. Minced Fancy diet given.

September 31st. Put on ordinary diet. Discharged on September 24th and could not be traced in November 1912.

Case/

Case 4. H. Y. Aet. 22. Female. Domestic Servant.  
Treated from May 31st to July 15th 1909. Gave a history of having had "indigestion" for several years. During the last two months has had epigastric pain immediately after taking food, which is relieved by vomiting. Has had slight haematemesis. "No meloena". Anaemic. Tongue furred. Foul breath. Teeth careous, especially the molars. Pressure over epigastrium elicited great tenderness especially over a point just above the umbilicus.

Treatment. Rest in bed.

May 31st. Rectal injections of  $\text{3vi}$  saline solution every four hours and  $\text{3ii}$  10% gelatin solution every two hours per oram.

June 3rd. Slight vomiting after gelatin therefore given it in 5% solution and no further vomiting.

June 8th. Rectal injections given every six hours and  $\text{3v}$  beef tea and two eggs given per oram.

June 15th. Injections stopped and 1 pint beef tea and egg custard given.

June 16th. Given Mist. Alba  $\text{3i}$  t.i.d.

June 29th. 4 oz. mince and 2 oz. potato included in diet and gelatin discontinued.

July 13th. Put on Fancy diet.

July 15th. Went home. Balcony treatment from July 2nd onwards. Complete rest in bed six weeks.

Weight June 14th, 5 st.  $8\frac{3}{4}$  lbs., July 5th 6 st.  $\frac{1}{2}$  lb.

November 1912. Seen and she stated that she had been quite well since discharge, able to eat anything and to follow her occupation.

Case/



Case 5. E. P. Aet. 23. Female. Packer.

Treated from February 15th to March 14th 1909.

History of pain in epigastrium sometimes after food.

Vomiting relieved the pain. Vomit consisted of partly digested food. No haematemesis and no meloena.

Recently has not been able to retain milk in stomach.

Always constipated. In-patient two years ago and treated for gastric ulcer with rest, milk diet and Bismuth. Physical signs very indefinite and only slight diffuse epigastric tenderness.

Treatment. Rest in bed.

February 16th. Rectal injections of  $\text{3v}$  saline solution every four hours and  $\text{3ii}$  10% gelatin solution every two hours per oram.

February 19th. Injections given only every six hours and  $\text{3i}$  beef tea and 1 egg given per oram twice a day.

February 25th. Injections discontinued and given a diet consisting of gelatin, 1 pint beef tea and 2 eggs.

March 1st. Gelatin stopped and 4 oz. mince and 2 oz. potato added to diet.

March 5th. Fancy diet given.

March 9th. Put on ordinary diet.

March 14th. Discharged cured.

Weight February 18th 6 st.  $7\frac{3}{4}$  lbs. February 29th

6 st.  $7\frac{1}{4}$  lbs. March 6th 6 st.  $10\frac{1}{2}$  lbs. March 15th

7 st.

Could not be traced in November 1912.

Case 6. M. O. Aet. 18. Female. Laundress.

Treated December 3rd, 1909 to January 21st, 1910.

Gave/

Gave off having for two months attacks of epigastric pain immediately after taking food, followed in from  $\frac{1}{2}$  - 1 hour by vomiting which relieved the pain. Has had a great deal of flatulence and troublesome constipation. Was under treatment at home for four weeks but relapsed and had slight haematemesis.

Anaemia. Teeth - molars carious. Localised and diffuse epigastric tenderness.

Treatment. Rest in bed.

December 3rd. Rectal injections of 3vi saline solution every four hours and 3ii gelatine solution every two hours per os.

December 12th. Injections discontinued and gelatin continued along with 1 pint of beef tea and 1 egg.

December 17th. Another pint of beef tea and another egg added.

December 20th. Mist. Alba 3i t.i.d.

December 22nd. Iron tonic given t.i.d.

December 24th. Gelatin discontinued and 4 oz. mince and 2 oz. potato added to diet.

December 30th. Full helping of mince given.

January 7th. Given minced ordinary diet.

January 10th. Put on ordinary diet.

January 21st. Discharged cured.

Weight. December 3rd, 7 st. 2 lbs. December 12th, 7 st.  $1\frac{1}{2}$  lbs. December 20th. 6 st.  $12\frac{1}{2}$  lbs.

December 28th, 7 st.  $\frac{1}{2}$  lb. January 3rd 7 st.  $3\frac{3}{4}$  lbs.

January 10th, 7 st. 5 lbs.

Could not be traced in November 1912.

Case/

Case 7. A. J. aet. 20. Female. Domestic servant.

Treated February 25th to April 15th, 1910. Gave a history of recurring attacks of epigastric pain, increased after taking food, during the past year. No vomiting nor haematemesis. For three weeks before admission had been on a milk diet without benefit.

Bowels regular. No meloena. Teeth carious. General tenderness over epigastrium with a localised area of inanimous intensity.

Treatment. Rest in bed.

February 25th. Rectal injections of  $\frac{3}{4}$  vi saline solution every four hours and  $\frac{3}{4}$  iii 10% gelatin solution per oram every two hours.

March 3rd. Injections discontinued and 2 pints beef tea and 2 eggs given along with the gelatin.

March 18th. Gelatin stopped and 4 oz. mince and 2 oz. potato added to diet.

March 22nd. One pint peptonised milk added.

March 29th. Given fancy diet.

April 12th. Put on ordinary diet.

April 15th. Discharged cured.

Treated on Balcony from March 28th onwards.

Weight. February 28th, 7 st.  $6\frac{1}{2}$  lbs. March 7th, 7 st.

$\frac{3}{4}$  lb. March 14th, 6 st. 13 lbs. March 21st, 7 st.

March 28th, 7 st.  $1\frac{1}{2}$  lbs. April 3rd, 7 st.  $6\frac{1}{2}$  lbs.

April 11th, 7 st.  $7\frac{3}{4}$  lbs. Rest in bed = 6 weeks.

Seen in December 1912. Since discharge has been quite well and able to take ordinary diet. Married.

Case/

Case 8. G. H. Aet. 36. Female. Domestic servant.

Treated from September 1st to November 11th, 1910.

History. Since the age of fifteen has had attacks of epigastric pain about half an hour after food. The pain was sometimes relieved by vomiting. In 1904 had a profuse haematemesis. Treated as an in-patient for gastric ulcer by rest in bed, milk diet with Bismuth and Soda in 1905 (twice), 1907, 1908 and was again admitted in 1908 when gastro-enterostomy was performed. This only gave temporary relief and pain and vomiting returned, and in July 1910 had profuse haematemesis. She was very anaemic. Teeth artificial. There was diffuse epigastric tenderness.

Treatment. September 1st - 9th, absolute rest in bed, milk diet along with Bismuth and Soda mixture t.i.d. and complained of epigastric pain and tenderness and vomited daily. Therefore on

September 9th. Given rectal injections of 3x saline solution every four hours and 3ii 10% gelatin solution per oram every 2 hours.

September 16th. Injections reduced to 3vi and given every 4 hours.

September 27th. Injections given every 8 hours and  $\frac{1}{2}$  pint of beef tea and 2 eggs given per oram along with the gelatin.

October 4th. Injections discontinued and 1 pint beef tea added.

October 14th. Gelatin stopped and 4 oz. mince and 2 oz. potato added to diet.

October 21st. Minced ordinary diet.

October 28th/



October 28th. Put on ordinary diet.

Rest in bed covered 57 days.

On October 4th had had no pain for 14 days but there was slight epigastric tenderness which cleared up by the 17th. Discharged on November 11th after having no pain after three weeks on ordinary diet.

Weight. September 2nd, 11 st. 11 lbs. September 19th, 7 st.  $3\frac{1}{2}$  lbs. September 26th, 7 st.  $1\frac{1}{4}$  lb. October 10th, 6 st.  $10\frac{1}{2}$  lbs. October 17th, 6 st.  $12\frac{1}{2}$  lbs. October 24th, 6 st.  $13\frac{3}{4}$  lbs. October 31st 7 st. 1 lb. November 10th, 7 st. 6 lbs.

Case 9. L. P. Aet 35. Female. Domestic servant.

Treated September 22nd to December 18th, 1910. Gave a history of eight months' duration of burning pain in epigastrium brought on by taking food. No vomiting nor haematemesis. Meloena. Under treatment greater part of time, including rest in bed and milk diet.

Constipated. Has had two previous similar attacks.

Teeth artificial. Localised area of tenderness in epigastrium with slight rigidity of left rectus.

Treatment. Rest in bed.

September 23rd. Put on rectal injections of  $\text{3viii}$  saline solution every four hours and  $\text{3ii}$  10% solution of gelatin every two hours per oram.

October 1st. One pint beef tea and one egg given per oram.

October 5th. Another pint of beef tea and another egg added.

October 12th/

October 12th. Injections discontinued and one oz. mince added.

November 1st. Gelatin stopped and 4 oz. mince and 2 oz. bread crumbs added to diet.

November 15th. Minced fancy diet given.

November 22nd. Put on ordinary diet.

December 18th. Discharged cured.

Rest in bed = 60 days.

Weight. September 22nd, 6 st.  $3\frac{1}{2}$  lbs. October 17th, 5 st. 9 lbs. November 21st, 6 st.  $1\frac{1}{4}$  lbs. December 13th, 6 st.  $4\frac{1}{2}$  lbs.

December 1912. Seen. Stated she had been quite well since discharge, taking ordinary diet, working.

Case 10. J. Mc. Aet. 38. Male. Labourer.

Treated from February 8th to March 24th 1910.

History extending over eight or nine years of epigastric pain about one hour after meals. Vomiting later which relieved the pain. Constipated as a rule. Has noticed that stools were black for the last few days and had a profuse haematemesis the night before admission. On admission complained of nothing and had no objective signs. Tongue clean and teeth good.

Treatment. Rest in bed.

February 8th. Given rectal injections of  $\text{3vi}$  saline solution every 4 hours and  $\text{3ii}$  10% gelatin solution every 2 hours per oram.

February 15th. Injections discontinued and 1 pint beef tea and 2 eggs given per oram.

February 25th./

February 25th. Gelatin stopped and another pint beef tea and 4 oz. mince and 2 oz. potato added to diet.

March 1st. Given Ferri et Ammon. Cit. grs X, Sod. Bicarb. grs X, Tr. Nuc. Vom. Mv, Aq. Chlorof. ad.  $3i$  t.i.d.

March 4th. Allowed custard.

March 11th. Fancy diet.

March 21st. Given ordinary diet.

March 24th. Discharged. Complete rest = 36 days.

Weight. February 28th, 9 st.  $5\frac{1}{2}$  lbs. March 14th, 9 st.  $14\frac{1}{2}$  lbs. November 1912, could not be traced.

Case 11. C.PL. Aet. 25. Female. Domestic.

Treated from January 6th to February 17th 1911.

Gave a history of frequent attacks during last six weeks of epigastric pain about  $\frac{1}{4}$  -  $\frac{1}{2}$  hour after taking food, followed later by vomiting which relieved the pain. No haematemesis nor meloena. Constipated. Teeth carious - most of the molars missing. Tongue dirty. Localised tenderness in epigastrium.

Treatment. Rest in bed.

January 6th. Injections of  $3vi$  saline solution given per rectum every 4 hours and also  $3ii$  10% gelatin solution every 2 hours per oram.

January 12th. One pint gelatin and one egg given by mouth.

January 16th. Injections discontinued and another pint beef tea and another egg added to diet.

January 19th. Gelatin stopped and 4 oz. mince and 2 oz. potato added to diet.

January 20th/

January 20th. One pint peptonised milk given.

January 27th. Minced fancy diet, and Balcony.

January 31st. Put on fancy diet.

During the first three days of treatment had occasional vomiting but this ceased and progress was uninterrupted.

Complete rest - 29 days.

Weight. January 10th, 6 st.  $13\frac{1}{2}$  lbs. January 17th, 6 st. 13 lbs. January 24th, 7 st.  $\frac{1}{2}$  lb. January 31st, 7 st. 3 lbs. February 7th, 7 st. 9 lbs. February 14th, 8 st.

November 1912. Seen and stated she had since discharge been quite well, taking ordinary diet and working.

Case 12. M. W. Aet. 26. Female. Domestic.

Treated from March 30th to May 9th, 1911.

History of seven weeks duration of attacks of pain - epigastric - coming on immediately after taking food and followed and relieved by vomiting. Vomit consisted of food. No haematemesis nor meloena. Patient very thin and pale. Teeth good. Slight localised epigastric tenderness.

Treatment. Rest in bed.

March 31st. Rectal injections of  $\text{3x}$  saline solution every four hours and  $\text{3ii}$  10% gelatin solution every two hours by the mouth.

April 7th. One pint beef tea and one egg allowed per oam. Given  $\text{3I}$  Mist. Alba t.i.d.

April 11th. Injections discontinued and given another pint of beef tea and another egg and one pint peptonised milk along with previous diet.

April 18th/



April 18th. Gelatin stopped and 4 oz. mince and 2 oz. potato added to diet.

April 26th. Minced ordinary diet.

May 2nd. Given ordinary diet.

May 9th. Discharged cured. Uninterrupted recovery.

Complete rest - 30 days.

Weight. March 31st, 5 st. 7 lbs. April 6th, 5 st. 6 lbs.

April 13th, 5 st. 8 lbs. April 25th, 5 st. 11 lbs.

May 2nd, 6 st.  $3\frac{1}{2}$  lbs.

Could not be traced in December 1912.

Case 13. N. O'L. Aet. 21. Female. Nurse.

Treated October 2nd to November 25th 1911. Gave a history of a heavy sensation behind sternum for past week after eating, with loss of appetite. Three days ago had a profuse haematemesis which was repeated the following day. Constipated. Tongue furred. Teeth - one or two with cavities. Slight localised epigastric tenderness. Meloena.

Treatment. Rest in bed.

October 2nd. Put on rectal injections of  $\frac{3}{4}$  vi saline solution every four hours and also  $\frac{3}{4}$  ii 10% gelatin solution every two hours per oram and 20 c.c. normal horse serum by the mouth as complained of excessive pain.

October 5th. Gelatin given in 5% solution.

October 6th. 20 c.c. normal horse serum given per oram.

October 10th. Given half pint of beef tea.

October 13th. Injections discontinued and given  $\frac{3}{4}$  ii 10% gelatin solution every 2 hours and two pints beef tea and 2 eggs by mouth.

October 17th/

October 17th. One pint peptonised milk added to diet.

October 25th. Gelatin stopped and 2 oz. mince added.

October 31st. Given porridge.

November 3rd. Allowed 4 oz. mince and 2 oz. potato.

November 10th. Put on fancy diet.

November 15th. Put on ordinary diet.

Constipation treated with 3i Mist. Alba t.i.d.

Vomited and complained of pain on October 3rd and 6th.

Complete rest - 28 days.

Weight. October 10th, 8 st. October 17th, 7 st. 12 lbs.

October 24th, 7.st. 12 lbs. October 31st, 8 st.

November 6th, 8 st. 2 lbs. November 13th, 8 st. 3 lbs.

November 20th, 8 st. 5½ lbs.

Seen in November 1912. Quite well since discharge, taking ordinary diet and working.

Case 14. F.L. Aet. 30. Female. Packer.

Treated September 14th to November 16th, 1911.

Gave a history extending over eleven years of occasional attacks of epigastric pain which passed through to back below left shoulder blade. Pain came on as a rule from one to two hours after taking food and was relieved by vomiting. Vomit consisted of partly digested food. Recently the attacks have been more frequent and severe. No haematemesis nor meloena. Constipated. Has been on milk diet for over a month and has lost weight.

General condition poor. Tongue furred. Teeth bad - upper molars carious. Localised epigastric tenderness.

Treatment/

Treatment. Rest in bed.

September 22nd. Rectal injections of  $\text{3viii}$  saline solution every four hours and also  $\text{3ii}$  10% gelatin solution per oram every 2 hours.

October 2nd. Given one pint beef tea and two eggs by the mouth.

October 10th. Injections stopped and another pint beef tea given.

October 20th. One pint peptonised milk allowed.

October 26th. Gelatin stopped and 4 oz. mince and 2 oz. potato added to diet.

November 1st. Full helping of mince allowed.

November 3rd. Put on minced ordinary diet.

November 16th. Discharged.

Weight. September 27th, 6 st. 11 lbs. October 11th, 6 st.  $6\frac{1}{2}$  lbs. October 18th, 6 st.  $3\frac{1}{4}$  lbs. October 24th, 6 st.  $2\frac{1}{4}$  lbs. November 1st, 6 st.  $1\frac{1}{2}$  lbs.

November 8th, 5 st.  $13\frac{1}{2}$  lbs. This patient's general condition was most unsatisfactory and there was a question of tuberculosis although there was no direct evidence of the disease. So far as gastric symptoms were concerned these quite cleared up and when seen in December 1912, she had had no return of them.

Case 15. E. B. Aet. 23. Female. Domestic servant.

Treated from January 31st to March 17th, 1911.

During the last two years has had recurrent attacks of epigastric pain coming on either immediately or within one hour after taking food. Occasional vomiting which relieved the pain. No haematemesis nor meloena.

Bowels/

Bowels regular. General condition fair. Anaemic.

Teeth - molars carious. Localised tenderness in epigastrium.

Treatment. Rest in bed.

February 3rd. Rectal injections of 3x saline solution every four hours and also 3ii 10% gelatin solution every two hours per oram.

February 10th. One pint beef tea and two eggs allowed.

February 14th. Injections discontinued and another pint of beef tea given.

February 20th. Gelatin stopped.

February 26th. 4 oz. mince and 2 oz. potato added to diet.

March 3rd. Put on minced fancy diet.

March 10th. Given ordinary diet and iron tonic t.i.d.

February 21st onward. Balcony treatment.

Weight. January 31st, 8 st. 10 $\frac{1}{2}$  lbs. February 7th, 8 st. 6 lbs. February 14th, 8 st. February 21st 8 st. 2 lbs. February 28th, 8 st. 5 lbs. March 5th, 8 st. 8 lbs. March 12th, 8 st. 11 lbs. March 17th, Discharged cured.

Seen in November 1912. Quite well since discharge, taking ordinary diet and working.

Case 16. M. B. Aet. 31. Female. Housewife.

Treated from March 15th to May 4th, 1911. For five months has had attacks of pain in the epigastrium - butning in character - coming on within one hour after food. No haematemesis nor meloena. Occasional vomiting of food which promptly relieves the pain.

Treated/



Treated three years ago for gastric ulcer - rest in bed, milk diet, Bismuth and Soda mixture and again seven months before this attack. Anaemia. Teeth artificial. Localised tenderness in epigastrium.

Treatment. Rest in bed.

March 17th. Rectal injections of 3 x saline solution every four hours and also 3ii 10% gelatin every two hours per oram.

March 28th. Given one pint beef tea and two eggs.

April 4th. Injections discontinued and one pint peptonised milk added to diet.

April 7th. Off gelatin and given 4 oz. mince and 2 oz. potato.

April 14th. Allowed full ration of mince.

April 21st. Given fancy diet.

April 28th. Given ordinary diet.

May 4th. Discharged cured.

Weight. March 15th, 6 st. 8 lbs. March 21st, 6 st.

3½ lbs. March 28th, 6 st. ½ lb. April 11th, 6 st.

½ lb. April 18th, 6 st. 2 lbs. April 25th, 6 st. 6 lbs.

Treated on Balcony from April 11th onwards. Seen in November 1912 and had been taking ordinary diet and quite well since discharge.

Case 17. G. A. Aet. 42. Female. Housewife.

Treated from March 30th to May 9th 1911. Gave a history of "indigestion" for seven or eight years.

During the last eight weeks has had frequent attacks of severe epigastric pain coming on immediately after food/

food and "shooting" through to the back. Vomiting which partly relieved the pain. A fortnight ago vomited half a pint of blood and noticed that stools were black. Generally constipated. General condition poor. Teeth - many wanting and those left carious. Localised tenderness in epigastrium over left rectus.

Treatment. Rest in bed.

March 31st. Given rectal injections of 3x saline solution every four hours and also 3ii 10% gelatin solution every two hours by the mouth.

April 10th. Allowed one pint beef tea and two eggs.

April 13th. Injections stopped and another pint beef tea added.

April 18th. Gelatin stopped and 4 oz. mince and 2 oz. potato added to diet.

April 28th. Allowed ordinary diet.

May 9th. Discharged cured.

Complete rest for 30 days.

Weight. March 30th, 6 st. 10 lbs. April 4th, 6 st.

10 lbs. April 11th, 6 st. 8 lbs. April 18th, 6 st.

4 lbs. April 25th, 6 st. 3 lbs. May 2nd, 6 st. 7½ lbs.

May 8th, 6 st. 10 lbs.

Could not be traced in December 1912.

Case 18. E. H. Aet. 26. Female. Teacher.

Treated from November 2nd to December 19th 1911.

Gave a history of "indigestion" for several years.

During the last month has had attacks of severe pain in the epigastrium immediately after food, which were relieved by vomiting. No haematemesis nor meloena.

Constipated. Anaemic. Dirty tongue. Teeth

artificial/

artificial. Localised epigastric tenderness.

Treatment. Rest in bed.

November 2nd. Given rectal injections of  $3\text{viii}$  saline solution every four hours and  $3\text{ii}$  10% gelatin solution every two hours per oram, and also 10 c.c. normal horse serum twice a day.

November 10th. Injections given every six hours and  $3\text{x}$  beef tea and two eggs.

November 12th. Complained of pain therefore eggs and beef tea stopped. 10 c.c. normal horse serum given b.i.d.

November 14th. No pain since the 12th. Injections given every four hours.

November 17th.  $3\text{x}$  beef tea, two eggs and Valentine's meat juice allowed.

November 19th. Injections discontinued.

November 21st. One pint peptonised milk added to diet.

November 27th. Another pint beef tea given.

December 1st. Gelatin stopped and 4 oz. mince and 2 oz. potato allowed.

December 8th. Given fancy diet.

December 12th. Given ordinary diet.

December 19th. Discharged cured. Complete rest - 43 days.

Weight. November 21st, 5 st.  $9\frac{1}{2}$  lbs. November 28th,

5 st. 6 lbs. December 5th, 5 st. 7 lbs. December

12th, 5 st.  $8\frac{1}{2}$  lbs. December 18th, 5 st. 10 lbs.

Seen in November 1912. Has been quite well since discharge, taking ordinary diet and teaching.

Case 19. G. B. Aet. 30. Male. Presser.

Treated from January 27th to March 11th 1911.

Gave/

Gave a history of three months duration of epigastric pain which was paroxysmal, about one hour after meals and occasional vomiting which gave relief. No haematemesis. Bowels regular. Anaemic. Teeth bad - molars deficient. Slight localised tenderness in epigastrium. Slight meloena.

Treatment. Absolute rest in bed.

January 27th - 31st. Given ordinary diet and complained of pain.

January 31st. Put on rectal injections of 3x saline solution every four hours and also 3 ii 10% gelatin solution every two hours per oram.

February 7th. Has had no pain for four days. Allowed one pint beef tea and two eggs.

February 10th. Injections discontinued and another pint of beef tea given.

February 17th. One pint peptonised milk added to diet.

February 20th. Gelatin stopped and 4 oz. mince and bread crumbs allowed. For two days after this complained of slight pain which then cleared up.

February 28th. Minced ordinary diet given.

March 7th. Put on ordinary diet.

Complete rest, 36 days.

I saw him in December 1912 and found he had a growth in the rectum, probably malignant.

Case 20. J. C. Aet. 30. Male. Labourer.

Treated from May 12th to June 25th 1911. Gave a history of having had for seven months attacks of epigastric pain coming on immediately after food, relieved/



relieved by vomiting some time later. No haematemesis nor meloena. Constipated. General condition poor. Teeth - molars carious. Localised area of tenderness in middle line in epigastrium.

Treatment. Rest in bed.

May 12th. Put on rectal injections of 3viii saline solution every four hours and also 3ii 10% gelatin solution every two hours per oram.

May 19th. Taken off injections and gelatin and given 4 oz. mince and 2 oz. potato and one pint peptonised mild.

June 6th. Minced ordinary diet.

June 9th. Ordinary diet given.

Complete rest, 32 days.

Weight. May 12th, 9 st. May 19th, 8 st. 12 lbs.

May 26th, 8 st. 10 $\frac{1}{2}$  lbs. June 1st, 9 st. 2 $\frac{3}{4}$  lbs.

June 10th, 9 st 5 lbs. June 17th, 9 st. 9 $\frac{1}{2}$  lbs.

Treated on Balcony June 10th onwards. Readmitted on September 4th and treated until October 5th, with a history of pain and vomiting after food. No objective signs. Treatment repeated.

Could not be traced in November 1912.

Case 21. E.C. Aet. 14. Male. Schoolboy.

Treated from October 10th to December 1st 1911.

Three weeks before admission had a sudden haematemesis and again a week later. Has no pain. Constipated.

Meloena. Very anaemic and general condition poor.

No physical signs.

Treatment/

Treatment. Absolute rest in bed.

- October 10th. Put on rectal injections of  $\frac{3}{vi}$  saline solution every four hours and  $\frac{3}{iii}$  10% gelatine solution every two hours per oram.
- October 17th. Injections discontinued and one pint beef tea and two eggs given.
- October 24th. Gelatin stopped and another pint of beef tea allowed.
- October 27th. Fancy diet and Iron and Arsenic mixture t.i.d.
- November 15th. Given ordinary diet. Complete rest 45 days.

Seen in December 1912. General condition greatly improved, and has been quite well since discharge.

Case 22. E. L. Aet. 49. Female. Cooper.

Treated from March 25th to May 2nd 1912.

Gave a history of having attacks of epigastric pain after taking food during the past two years. Pain came on about half an hour after meals. No vomiting. Constipated. Tongue furred. Teeth artificial. Slight localised tenderness in epigastrium.

Treatment. Absolute rest in bed.

- March 25th. Given rectal injections of  $\frac{3}{viii}$  saline solution every four hours and  $\frac{3}{ii}$  10% solution of gelatin every two hours per oram.
- April 2nd. Allowed one pint beef tea and one egg.
- April 6th. Injections discontinued and another pint of beef tea and another egg allowed.
- April 13th. 4 oz. mince and 2 oz. potato added to diet.
- April 23rd. Gelatin stopped and given minced ordinary diet.
- April 28th/

April 28th. Put on ordinary diet.

Complete rest, 28 days.

Seen in November 1912 and has no symptoms and has been taking ordinary diet since discharge.

Case 23. E. C. Aet. 20. Female. Servant.

Treated from May 14th to June 28th 1912.

History of attacks of epigastric pain for four months coming on about half an hour after taking food.

Also pain which "went through to back". Pain was followed by vomiting which gave relief. One month ago vomited half a pint of blood and also had meloena. Constipated. Has been under treatment at home.

Slightly anaemic. Tongue furred. Teeth - several carious. Tenderness localised in left hypochondrium and also slight resistance of left rectus.

Treatment. Rest in bed.

May 14th. Given rectal injections of 3viii saline solution every four hours and also 3ii 10% gelatin solution every two hours per oram.

May 21st. Injections discontinued and one pint beef tea and two eggs allowed per oram.

May 28th. Another pint beef tea allowed.

May 31st. Gelatin stopped and 4 oz. mince and 2 oz. potato added to diet.

June 4th. Given full ration of mince.

June 11th. Put on minced ordinary diet.

June 18th. Given ordinary diet.

Weight. May 14th, 7 st. 11 $\frac{1}{2}$  lbs. May 21st, 7 st. 8 $\frac{1}{4}$  lbs.

May/

May 28th, 7 st.  $5\frac{1}{2}$  lbs. June 1st, 7 st.  $5\frac{3}{4}$  lbs.  
 June 8th, 7 st.  $6\frac{3}{4}$  lbs. June 15th, 7 st. 11 lbs.  
 June 25th, 8 st. 1 lbs.

Complete rest for 38 days.

Case 24. M. F. Aet. 25. Female. Charwoman.

Treated April 6th to May 18th 1912.

Three weeks history of epigastric pain coming on about ten minutes after taking food. Vomiting which relieved the pain. Slight haematemesis. Bowels regular. Has had several previous attacks. Anaemic. Teeth - upper artificial and lower carious. Localised tenderness in middle line in epigastrium.

Treatment. Rest in bed.

April 7th. Given rectal injections of  $\text{3viii}$  saline solution every four hours and also  $\text{3ii}$  10% gelatin solution every two hours per oram.

April 12th. Injections stopped and allowed one pint beef tea and two eggs per oram.

April 18th. Another pint of beef tea given.

April 28th. Gelatin stopped and 4 oz. mince and 2 oz. potato added to diet.

April 30th. Minced ordinary diet allowed and given Ferri et Ammon. Cit. grs.x and Mist. Alba  $\text{3i}$  t.i.d.

May 14th. Put on ordinary diet.

Weight. April 6th, 7 st. 13 lbs. April 16th, 7 st.  $4\frac{3}{4}$  lbs. April 23rd, 7 st.  $3\frac{1}{2}$  lbs. April 30th, 7 st.  $3\frac{3}{4}$  lbs. May 7th, 7 st. 4 lbs. May 14th, 7 st.  $10\frac{5}{4}$  lbs.

Complete rest 34 days. Not traced in December 1912.

Case/



Case 25. R. M. Aet. 23. Female. Dressmaker.

Treated from March 21st to May 19th 1912.

Gave a two years' history of recurring attacks of epigastric pain and also pain in the back immediately after food and partially relieved by vomiting. Slight hæmatemesis seven months ago. Since then vomit has had "coffee grounds" appearance. Constipated - motions "dark" in colour. Slightly anaemic. Teeth - molars carious. Localised tenderness in epigastrium.

Treatment. Rest in bed.

March 21st. Given rectal injections of  $\frac{3}{4}$  viii saline solution every four hours and also  $\frac{3}{4}$  ii 10% gelatin solution every two hours per oram.

March 29th. Allowed one pint beef tea and two eggs per oram.

April 2nd. Injections discontinued and another pint of beef tea allowed.

April 4th. One pint peptonised milk given.

April 9th. Gelatin stopped and 4 oz. mince and 2 oz. potato added to diet.

April 18th. Full ration of mince.

April 30th. Minced ordinary diet given.

May 11th. Put on ordinary diet.

Complete rest - 40 days.

Weight. March 26th, 9 st.  $3\frac{1}{2}$  lbs. April 2nd, 9 st. 2 lbs. April 16th, 9 st.  $\frac{3}{4}$  lb. April 23rd, 9 st.  $3\frac{1}{2}$  lbs. April 30th, 9 st.  $6\frac{1}{2}$  lbs. May 7th, 9 st. 8 lbs.

Before admission had had institutional and domiciliary treatment comprising rest, milk diet and a bismuth mixture/

mixture. Readmitted one month later complaining of pain after food and occasional vomiting. No physical signs. Treated on same lines as before.

Seen in December 1912 and had been taking ordinary diet with no return of symptoms since discharge.

Case 26. J. A. Aet. 36. Female. Housewife.

Treated February 12th to April 13th 1912.

Gave a history of "indigestion" for ten or twelve years with recurring attacks of epigastric pain from 1 - 1½ hours after meals. Vomiting relieved the pain.

Recently the attacks have been more severe and frequent.

Five weeks ago had a severe haematemesis (four pints) and this was repeated one week ago. Constipated.

Meloena. Blanched appearance. Teeth fairly good.

Localised tenderness in epigastrium.

Treatment. Absolute rest in bed.

February 12th. Given rectal injections of 3viii saline solution every four hours and also 3ii 10% gelatin solution every two hours per oram.

February 22nd. One pint beef tea and two eggs allowed.

February 27th. Injections discontinued and another pint of beef tea allowed.

March 5th. Gelatin stopped and 4 oz. mince and 2 oz. potato added to diet.

March 12th. Given full ration of mince.

March 15th. Put on minced ordinary diet.

March 19th. Given ordinary diet.

Complete rest - 52 days.

Case/

Case 27. L. W. Aet. 28. Female.

Treated January 24th to March 11th.

History. For the last seven years has had frequent attacks of epigastric pain. These came on after food and lasted for a variable time, being relieved by vomiting. Present attack began two months ago. No haematemesis and no meloena. Constipation. Has been under treatment - rest in bed, milk diet and Bismuth - but without relief. Anaemic. Teeth fairly good. Slight localised tenderness in epigastrium.

Treatment. Rest in bed.

January 24th. Given rectal injections of  $\frac{3}{4}$  viii saline solution every four hours and also  $\frac{3}{4}$  iii 10% gelatin solution every two hours per oram.

February 1st. Allowed one pint beef tea and two eggs.

February 5th. Injections discontinued and given another pint of beef tea.

February 6th. Allowed two pints peptonised milk.

February 12th. Gelatin stopped and 4 oz. mince and 2 oz. potato added to diet.

February 19th. Minced ordinary diet.

February 27th. Given ordinary diet.

February 6th onwards - treated on Balcony.

Weight. February 13th, 8 st.  $1\frac{1}{2}$  lbs. February 20th, 8 st.  $4\frac{1}{2}$  lbs. February 27th, 8 st.  $6\frac{1}{4}$  lbs. March 5th, 8 st. 11 lbs. Complete rest - 39 days.

Seen in December 1912 and had been taking ordinary diet since discharge with no return of symptoms.

Case/

Case 28. E. W. Aet. 21. Female. Domestic servant.

Treated from February 12th to March 29th 1912.

Gave a history of repeated attacks of severe epigastric pain for the last six weeks. The pain comes on soon after taking food and is relieved by vomiting which occurs about one hour later. No haematemesis and no meloena. Bowels regular. Tongue furred. Teeth - molars decayed stumps. Localised tenderness to left middle line in epigastrium.

Treatment. Absolute rest in bed.

February 12th. Given rectal injections of 3viii saline solution every four hours and also 3ii 10% gelatin solution every two hours per oram.

February 20th. Half a pint of beef tea allowed.

February 22nd. Given one pint of beef tea and one egg.

February 27th. Injections stopped and another pint beef tea and another egg given.

March 2nd. Gelatin stopped and 4 oz. mince and 2 oz. potato added to diet.

March 9th. Put on minced ordinary diet.

March 12th. Given ordinary diet.

Absolute rest for 33 days.

Seen in December 1912, was quite well and able to eat anything. Married.

Case 29. E. S. Aet. 28. Female. Domestic servant.

Treated from January 20th to March 8th. Gave a history of frequent attacks of epigastric pain for the last three months. The pain came on immediately after taking/



taking food and was also referred to left shoulder. Vomiting gave relief. Slight haematemesis one week ago. Constipated. Has had several previous attacks. Anaemic. Teeth - molars decayed. Localised tenderness in the epigastrium.

Treatment. Rest in bed.

January 20th. Given rectal injections of saline solution every four hours also 3 ii 10% gelatin solution every two hours per oram.

February 1st. Allowed one pint beef tea and one egg.

February 5th. Injections discontinued and another pint of beef tea and another egg added to diet.

February 9th. One pint peptonised milk added.

February 14th. Gelatin stopped and 4 oz. mince and 2 oz. potato given.

February 20th. Full ration of mince.

February 23rd. Minced fancy diet given.

February 29th. Put on ordinary diet.

Complete rest for 44 days.

Weight. February 13th, 7 st. 12 lbs. February 20th, 8 st. 2 lbs. February 27th, 8 st. 5 $\frac{3}{4}$  lbs. March 5th, 8 st. 7 lbs.

Seen in December 1912. Quite well, taking ordinary diet and working as a domestic servant.

Summary of Treatment.

Rest in bed is essential. All sources of septic infection should be removed. In every case in which it was necessary carious teeth were extracted and the mouth kept sweet with a frequent mouth wash of Hydrogen Peroxide.

Rectal injections of saline solution -giving from six to ten ounces at each injection every four hours - were given on an average for a period of ten days. In every case these were well retained and in no case was there any rectal irritation.

Gelatin was given in 10% solution and in a few cases 5% solution. Two ounces were given every two hours. It was either given alone or flavoured with lemon and later sometimes in beef tea.

Beef tea was given after the first week. In twenty four hours one pint was given at first and later increased to two pints. It was given in quantities of five ounces at a time.

Eggs were given beaten up raw.

The mince was given very lightly cooked.

Peptonised milk was given to those patients who were treated on the Balcony in the open air.

Fancy diet, white flesh only, along with farinaceous articles of diet, coffee and cocoa.

In the above series of cases of gastric ulcer

44.8% gave a history of haematemesis. Only six patients, or just over 20%, had sound teeth, five patients had artificial teeth and eighteen had teeth in a more or less carious condition.

Constipation was a prominent feature and occurred in over 68% of the cases.

#### Result of treatment.

In every case the result of the treatment was most satisfactory and during its whole course I did not have a single complaint of any sort from a patient. They were comfortable and contented and did not suffer from thirst or the pangs of hunger. The general condition of the patients was well maintained while on gelatin and rectal injections, and in almost every case the general appearance rapidly improved during its course. There was only a very slight and practically negligible loss of weight during the early part of the treatment whilst on gelatin and rectal saline. The average decrease in weight, in nineteen cases, which were weighed and which were on rectal injections and gelatin for periods of ten days and twenty-one days respectively, was four and a half pounds. In one case there was a steady loss of weight throughout as recorded. With this exception the decrease was very quickly made up or exceeded. This was/

was especially marked when the latter part of the treatment was carried out in the open air on the Balcony. In many instances the change in the general appearance and condition of the patients so treated was very striking.

Coming now to the effect of the treatment on the symptoms - Pain was quickly relieved in the majority of cases and only in three cases was it necessary to resort to other measures for the relief of this symptom. As a rule all pain had quite cleared up by the second or third day and in those cases in which it did not respond to the gelatin, the administration of 10 - 20 c.c. of normal horse serum two or three times a day by the mouth, as recommended by Hört (30), was found to be a most satisfactory analgesic. Vomiting was never troublesome and practically never recurred after treatment was established. Although in 44.8% of the cases there was a history of haematemesis, the majority being recent, in no case was there the slightest indication for delaying the administration of gelatin by the mouth and in no case was there a recurrence of the haemorrhage.

In November and December of 1912 I tried to visit all of the patients, but found it impossible to trace many of them. However, I saw fifteen and of these all were quite well as regarded any gastric condition.

In/



In conclusion, I claim that by the administration of gelatin by the mouth and rectal alimentation by means of saline solution, the general condition of the patient can be maintained in a satisfactory state; that symptoms are quickly relieved; that the comfort of the patient is assured; and that the healing process is accelerated. By following the dietary, the patient is brought to a state of tolerance for ordinary food and there is no necessity for him after treatment is completed to beware of ordinary home fare, except in excessive quantities.

As regards nursing, the Sisters of my male and female wards have both repeatedly stated that the patients are more contented and easier to manage, and that the carrying out of the routine treatment is no more irksome than any other method of treatment.

In recommending this method of treatment, I have endeavoured that my line of thought should be suggestive rather than dogmatic. In reviewing the most recent knowledge, I have sought to make clear the difference in treatment and to ask myself - Are we the gainers by the change? I think we may safely say that we can recognise considerable additions to our knowledge and, as a distinguished author has said, "It is ever thus with the advancing tide of scientific research which inaugurates new systems as the old ones ripen and die out".

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